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HEAD INJURY

A STUDY OF PATIENTS WITH CHRONIC POST-TRAUMATIC COMPLAINTS

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The resemblance from case to case of certain sequelae to injuries of the head has led to a tendency to group these symptoms together in syndromes and to forget the patient behind them. These syndromes have been variously labeled—postconcussion syndrome, postcontusional state, post-traumatic meningeal headache, post-traumatic circulatory instability and traumatic neurosis—according to the inferred cause, and various rules of thumb have been suggested for their handling. In actual fact, there is no such thing as "post-traumatic headache" in the abstract. There are only individual patients complaining after injury to the head of headache, dizziness, fatigue, irritability, "nervousness," impaired efficiency or other mental symptoms. In view of the fact that few complaints other than these could be made by a person who finds himself in an unsatisfactory situation after such an injury, it is evident that a superficial uniformity of symptoms may cover a variety of physical disorders and disturbances in personality. We report here the study, by both physical and psychologic methods, of a series of patients with such complaints, in an effort to sort out the various factors which are associated with them. A thorough review of the literature up to 1942 has been presented by Denny-Brown.[1] We shall refer to only a few of the recent observations elsewhere, together with the data obtained from the study of patients in this clinic.

MATERIAL AND METHODS

The material on which our data are based includes the results of direct studies by neurologic, psychiatric, physiologic, psychologic and, in some cases, neurosurgical means[2] on 90 subjects several months or longer after an injury to the head, the series including 15 patients studied at repeated intervals during recovery from such injury, and the results of a follow-up inquiry into the effects of subdural insufflation and other methods which were used in this institute in the treatment of 82 other patients with post-traumatic headache or dizziness. The 90 subjects included patients, both civilian and military, with and without compensation claims, who were referred because of their symptoms, and several control patients, some with symptoms and some without, who returned for study at our request at a time after the injury comparable to that for the patients, but who would not otherwise have sought medical care.

The data on these 90 subjects are summarized in table 1, of which the main body of this paper is an explanation, under the following headings: (1) types of headache and controls, (2) previous personality background, (3) severity of injury, (4) electroencephalographic and pneumoencephalographic ratings, (5) results of the Rorschach test, (6) situational factors and (7) summary of significant associations and trends. The data on the series of 82 patients given insufflation treatments are presented in section 8. We shall also discuss (9) the vascular responses to posture, and, finally, we shall present a concept of (10) mechanisms, with implications for management.

I. TYPES OF HEADACHE AND CONTROLS

In spite of the superficial uniformity of complaints already mentioned, it has been our experience that a careful inquiry into to what the patient means by his complaint, such as headache or dizziness, will give a valuable lead to the solution of the problem. The kind of headache has been particularly useful in this respect and has provided a basis for classification into several types. The relative importance of physical and psychologic factors in the various types will be discussed later.

The following types of headache are described: (1) localized, (2) hemicranial, (3) occipital, (4) bilateral, (5) generalized and (6) bizarre. There is shading between these types, and some patients will describe a headache which might

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1. Denny-Brown, D.: Sequelae of War Head Injuries, New England J. Med. **227**:771-780 (Nov. 19); 813-821 (Nov. 26) 1942.

2. The neurosurgical staff, under Dr. W. Penfield, Dr. W. V. Cone and Dr. A. R. Elvidge, carried out the surgical procedures; Dr. A. E. Childe made the roentgenographic investigations, and Dr. H. H. Jasper, the electroencephalographic studies.

TABLE 1.—*Data on Ninety Patients with Head Injury**

Condition	Total No.	Unstable Personality Background			Severity of Injury				Electroencephalographic Ratings				Mean Time After Injury	Pneumoencephalographic Ratings				Rorschach Ratings: Instability Mean	Rorschach Ratings: Instability Standard Error	Rorschach Ratings: Disability Mean	Rorschach Ratings: Disability Standard Error	Situational Factors: None	Situational Factors: Compensation (A) (B)	Situational Factors: Military Service	Situational Factors: Draft	Situational Factors: Legal Factors	Situational Factors: Individual Problems
		0	1	2	0	1	2	3	4	0	1	2	3		0	1	2	3	4									
Headache																												
Localized	22	**18**	**3**	**0**	**2**	**8**	**6**	**2**	**3**	**8**	**4**	**3**	**4**	1 yr. 5 mo.	**0**	**2**	**2**	**4**	**1**	**14.7**	± **1.69**	**13.1**	± **1.65**	**3**	**4**	**5.5**	**1.5**	**3**
Hemicranial	3	**0**	**3**	**0**	**1**	**2**	**2**	**0**	**0**	**2**	**1**	**0**	**0**	2 yr. 2 mo.	**1**	**0**	**2**	**0**	**0**	**8.0**	± **4.73**	**3.7**	± **2.18**	**2**	**0**	**0**	**0**	**1**
Occipital	11	**6**	**1**	**4**	**5**	**2**	**2**	**1**	**0**	**3**	**2**	**1**	**1**	2 yr. 6 mo.	**0**	**1**	**2**	**1**	**0**	**13.9**	± **3.28**	**8.3**	± **2.20**	**3**	**0**	**1**	**0**	**1**
Bilateral	13	**4**	**5**	**3**	**4**	**3**	**2**	**4**	**0**	**7**	**1**	**4**	**1**	1 yr. 8 mo.	**0**	**0**	**2**	**1**	**0**	**18.7**	± **3.36**	**9.1**	± **1.69**	**4**	**0**	**0**	**0**	**1**
Generalized	7	**1**	**3**	**3**	**4**	**0**	**2**	**1**	**0**	**4**	**0**	**2**	**1**	1 yr. 8 mo.	**0**	**0**	**2**	**1**	**0**	**17.0**	± **3.44**	**11.5**	± **3.31**	**2**	**0**	**2**	**2.5**	**0**
Bizarre	11	**2**	**7**	**2**	**2**	**6**	**1**	**2**	**0**	**6**	**4**	**1**	**0**	1 yr. 1 mo.	**0**	**0**	**2**	**3**	**1**	**20.1**	± **3.06**	**4.6**	± **2.10**	**2.5**	**2**	**0**	**2.5**	**2**
Other symptoms	9	**1**	**5**	**3**	**4**	**3**	**3**	**0**	**0**	**6**	**2**	**1**	**0**	1 yr. 3 mo.	**0**	**0**	**2**	**2**	**0**	**15.2**	± **3.64**	**5.4**	± **2.18**	**2**	**0**	**3**	**0**	**2**
Controls:																												
With some headache	8	**2**	**5**	**1**	**2**	**1**	**2**	**1**	**1**	**3**	**1**	**2**	**0**	1 yr.	**0**	**0**	**0**	**0**	**0**	**15.0**	± **3.62**	**6.9**	± **1.34**	**7†**	**0**	**0**	**0**	**0**
With no headache	11	**8**	**3**	**0**	**3**	**4**	**1**	**1**	**2**	**6**	**2**	**3**	**0**	1 yr.	**0**	**0**	**0**	**0**	**0**	**16.6**	± **3.42**	**4.1**	± **2.06**	**7†**	**3**	**0**	**0**	**1**
With evidence of organic defects	3	**3**	**0**	**0**	**0**	**0**	**1**	**1**	**1**	**1**	**0**	**1**	**1**	..... 6 mo.	**0**	**0**	**1**	**1**	**0**	**18.0**	± **16.0**	**13.5**	± **5.50**	**2†**	**1**	**0**	**0**	**0**
Total	90	**43**	**30**	**14**	**25**	**27**	**20**	**9**	**9**	**41**	**16**	**16**	**7**	1 yr. 4 mo.	**1**	**6**	**11**	**11**	**2**	**16.0**	± **0.92**	**8.5**	± **0.81**	**25**	**8**	**19**	**4**	**15**

* The figures in bold face are those which show a significant difference from the rest in the series.
† The significance for these figures was established by the combination of two or three groups.

be classified under either of one of two types, but most of the headaches are recognizable as conforming to one of the following patterns. 1. The pain of localized headache has its onset, or is maximal, in one area on one side of the head, with a variable amount of extension to the rest of that side of the head. The site of the pain is usually where the patient was struck, but it may be the region of a contrecoup injury. It is most frequently frontal or parietal. Some patients have almost constant pain, but most of them have intermittent bouts, varying from several a day to about one a month, usually brought on by fatigue or exertion, sometimes by damp weather, sometimes by emotional stress and frequently by stooping or sudden movements of the head, and usually relieved by rest. We have the impression that many headaches of this kind were continuous at first, when the pain was more severe, but became less frequent as recovery progressed. 2. It may be that the hemicranial type is at one end of the localized series and is a late development in some cases of that form, for our patients with hemicranial headache were seen, on an average, longer after the injury than were the patients with the localized type. The hemicranial headache was of sudden onset, lasted several hours to two or three days and occurred from once a week to once a month, on the whole resembling migraine headache. 3. Occipital or suboccipital headache is bilateral and is sometimes accompanied by tenderness at the attachment of the posterior muscles of the neck, as though due to tension in these muscles. Occipital headache may be accompanied by the frontal type, thus shading over into the next form. 4. Bilateral headache may be frontal, temporal or parietal. There may be generalized extension, but the patient describes the maximal pain or the onset of the attack as in corresponding areas on the two sides of the head. 5. In generalized headache the pain is "all over" the head, and there is no localization. 6. The bizarre type includes the feeling of pressure over the vertex, the sensation of a "tight band" around the head and the pain which wanders from one part of the head to another, either according to the patient's presenting description or as reported on different occasions. Sometimes what has been superficially reported as a "headache" has resolved itself on closer questioning into a "crackling" feeling in the head or some other bizarre sensation and we have classified it under this head.

The incidence of these types of headache in our series is shown in table 1, which also includes data on patients presenting chiefly symptoms other than headache and on three small

control groups. The control patients with headache were those who returned at our request, and not because of their symptoms, but who had had some headache since their injury. The figures for these patients have been included in the distribution according to the type of headache; so these figures are not included in the totals at the foot of the table. Of this small group of 22 patients whom we were able to bring back for study, 8 had had headaches but carried on with their work, 3 had evidence of frank "organic" effects of the injury and only 11 had been completely free from headaches or other symptoms after a reasonable period of convalescence. It was difficult to get a larger number of symptom-free controls among persons with former head injuries, as many either had moved away on military duty or, because of their freedom from symptoms, were not sufficiently interested to cooperate.

The relation of the other evidence to the type of headache will be considered after we have explained how each kind of evidence was obtained.

2. THE PREVIOUS PERSONALITY BACKGROUND

As an assessment of the previous stability of the patients and the controls during childhood and up to the time of the accident, we employed a psychiatric interview planned to obtain certain comparable data. This was based on the kind of data reported as frequently obtained from soldiers who failed to return to duty after a neurotic illness [3] and from many persons exhibiting neurotic reactions in wartime.[4] The points covered in every interview were as follows: (1) family history of nervous or mental disease, including neurosis, alcoholism and "sick headache"; (2) history of a broken home before midadolescence, death of a parent or divorce of, or serious quarreling between, parents; (3) previous history of nervous symptoms or emotional breakdown or of a psychosomatic disorder occurring in relation to stress; (4) presence of childhood fears, such as fear of the dark, requiring a night light, or night terrors; (5) enuresis after the age of 6 years; (6) nail biting after the age of 10; (7) tics of more than brief duration; (8) stammering; (9) sleep walking on more than three occasions; (10) poor school record, truancy or attainment of a grade two years over the average age; (11) avoidance of fights as a boy; (12) avoidance of dangerous games; (13) excessive reaction to interests, wakefulness at night before an exciting event, going off food for two or three meals, slight diarrhea or exhaustion after such an event; (14) light, restless sleep; (15) fainting at the sight of blood; (16) a hypochondriacal attitude, including total abstinence from drinking and smoking if for reasons of health; (17) poor work history, frequent changes in jobs or dissatisfied attitude toward work, and (18), if the patient was in one of the services, inadequate motivation, such as reasons other than a desire to fight or to do his part.

It is realized that these items include a mixture of factors predisposing to instability and of actual symptoms of neurosis and that there is only empiric justification for their being presented together. However, they have proved useful even in this unanalyzed way, without any attempt to determine their relative value and significance. Although their significance in any particular case must be considered in the light of clinical judgment, we believed that a more objective rating was possible if these points were used in a purely quantitative way.

The ratings for an unstable personality background, shown in table 1, were obtained by scoring 0 if none to two of the aforementioned eighteen items were present; 1, if three to four items were present, and 2, if five or more items were positive. Avoidance of fights and dangerous games was not counted in the rating for female patients. The numbers in the table opposite each type of headache and each group of controls represent the number of patients presenting each rating. It will be noted that the totals under "unstable personality background" do not quite reach 90. This discrepancy is present to varying degrees for the different types of evidence because, for one reason and another, every type of evidence could not be obtained for every subject studied.

Eight psychoneurotic patients, without head injury, were also studied according to the same plan of interview and by the same examiner who interviewed all the patients data on whom are recorded in table 1. Of these patients, 5 gave ratings of 2, for the item under discussion, and 3, ratings of 1. These subjects are too few to provide an adequate group of psychoneurotic controls, but similar data have frequently been reported in the histories of neurotic patients. It will be seen from the table that the controls with no headache, the controls with organic defects as a result of the accident and the patients with localized headache were low in evidence of a previous unstable personality background.

3. Lewis, A., and Slater, E.: Neurosis in Soldiers: Follow-Up Study, Lancet **1**:496-498 (April 25) 1942.

4. Gillespie, R. D.: Psychological Effects of War on Citizen and Soldier, New York, W. W. Norton & Company, Inc., 1942, p. 251.

The other patients tended to have personality backgrounds similar to those of the psychoneurotic patients.

3. SEVERITY OF INJURY

Each patient was rated with regard to severity of injury on a scale of 0 to 4. Criteria for this rating included the duration of post-traumatic amnesia, which has been considered as the greatest single prognostic criterion in cases of head injury[5] and, when the patient had been under observation in a hospital, as had been the case with the majority of patients with severe injuries, the amount of blood in the spinal fluid, the presence of fracture of the skull and the presence of neurologic signs.

A rating of 0, or negligible injury, indicated loss of consciousness for less than ten minutes and absence of fracture. A rating of 1 indicated either post-traumatic amnesia for ten to thirty minutes or the presence of fracture of the skull or of from 100 to 1,000 red cells per cubic millimeter in the spinal fluid. A rating of 2 indicated the presence of any two of the following features: Post-traumatic amnesia for one to three hours; fracture of the skull; 1,000 to 20,000 red cells per cubic millimeter in the spinal fluid, and slight neurologic signs. A rating of 3 indicated post-traumatic amnesia of from three to forty-eight hours' duration and fracture of the skull, a red blood cell count up to 100,000 per cubic millimeter or pronounced neurologic signs. A rating of 4, or very severe injury, indicated the presence of three of the following features: post-traumatic amnesia of over forty-eight hours, fracture of the skull, a red cell count of over 100,000 per cubic millimeter of spinal fluid and pronounced neurologic signs.

These criteria are described only as approximations, for they were essentially clinical ratings, which took into consideration possible distortions of the post-traumatic amnesia and discrepancies in various sources of information and other relevant data. They are given not as representing sharp lines of differentiation in the degrees of injury but as indicating roughly the severity of injury.

In table 1 the number of patients with each rating is charted in relation to each type of headache and each control group. It will be seen that the patients with organic defects and those with localized headache tended to have relatively more severe injuries, while patients with hemicranial headache, generalized and bizarre headache and symptoms other than headache tended to have less severe injuries.

4. ELECTROENCEPHALOGRAPHIC AND PNEUMOENCEPHALOGRAPHIC RATINGS

The next sections of table 1 present ratings for electroencephalographic and pneumoencephalographic abnormalities. For 80 patients we had the results of electroencephalographic examination at some time between three months and four years after injury or else a normal electroencephalogram made in less than three months after the accident, indicating that the pattern was still normal within this period. The mean time after injury at which the electroencephalographic study was made on each group is indicated in the table. For 31 of these patients we also had pneumoencephalographic studies. For comparison of the results of these methods of study with the other data, we had them rated independently of the clinical information. Dr. Jasper rated the electroencephalographic abnormalities on a 4 point scale. Dr. Childe rated the roentgenographic evidence, using a score of 0 to 3 for degrees of ventricular dilatation and a score of 0 to 2 for degrees of widening of the sulci. These two scores were added to give a 5 point rating, though no patient achieved such a score.

The relation of these ratings to the type of headache will be considered in section 7 of this paper.

As in the series reported by Williams,[6] about one-half our patients with chronic symptoms showed electroencephalographic abnormalities. However, the ratio was found to be the same for our control patients as for the patients who complained of symptoms.

Both the electroencephalographic and the pneumoencephalographic ratings showed a slight correlation with the severity of the injury: 0.264 ± 0.085 for 53 electroencephalographic ratings made between three months and two years after the injury and 0.383 ± 0.102 for 31 pneumoencephalographic ratings. We were not able to show a significant correlation between the electroencephalographic ratings obtained over two years after the injury and the severity of the injury, or between the electroencephalographic and the pneumoencephalographic ratings. However, no patients had a report of electroencephalographic abnormality which was not substantiated by pneumoencephalographic evidence when this was obtained also. In table 2

5. Jefferson, G.; Cairns, H.; Brain, W. R., and Guttmann, L., in Discussion on Rehabilitation After Injuries to the Central Nervous System, Proc. Roy. Soc. Med. **35**:295-308 (Feb.) 1942.

6. Williams, D.: The Electroencephalogram in Chronic Posttraumatic States, J. Neurol. & Psychiat. **4**:131-146 (April) 1941.

the patients with a high pneumoencephalographic rating and a low or negative electroencephalographic rating were those tested two years or more after the injury, when the electroencephalographic abnormality had diminished but the pneumoencephalographic abnormality remained. If the results of both procedures had been determined for more patients from six months to one year after injury, a significant positive correlation might have been demonstrable.

The demonstration in table 2 that the electroencephalographic ratings were usually more conservative than the pneumoencephalographic ratings, together with the relatively greater convenience of the electroencephalographic procedure than of the pneumoencephalographic method, indicates a considerable degree of usefulness for the electroencephalogram in the assessment of the status of patients with complaints after head injury, at least less than two years after the injury. When, however, there are medicolegal considerations or the question is one of compensation or pension, neither the electroencephalographic nor the roentgenographic evidence should be used without the severity of the injury and the constitution of the patient prior to the injury being considered. Either method might reflect not only the effects of the injury itself but a cerebral abnormality antedating the accident.

TABLE 2.—*Numbers of Cases in Relation to "Organic" Ratings*

		Pneumoencephalographic Rating					
		0	1	2	3	4	Totals
Electroencephalographic rating	0	1	1	3	2	2	9
	1	0	2	2	4	0	8
	2	0	1	3	4	0	8
	3	0	1	3	2	0	5
	4	0	0	1	0	0	1
Totals		1	5	11	12	2	31

5. RESULTS OF THE RORSCHACH TEST

Psychologic tests used in this study included the Binet and Snedden word lists,[7] the repetition of digits, the Kraepelin continuous subtraction test[8] and the Shipley-Hartford retreat test,[9] as well as the Rorschach method. The word lists did not differentiate the clinical types. Subjects without headache and patients with various types of headache showed mean verbal intelligence levels corresponding with intelligence quotients of about 100. The span of digits and the continuous subtraction test gave poor results with evidence of severe damage to the brain or of severe neurosis but did not appear to differentiate between the two conditions. The Shipley-Hartford test has been used to indicate the presence of subdural hematoma,[10] but it apparently does not measure lesser degrees of "organic" impairment, for its results in our series showed no relation to severity of the injury or to electroencephalographic or pneumoencephalographic evidence of cerebral damage except in cases of extreme injury.

The Rorschach method, on the other hand, gave striking results in a comparison of patients with head injury and patients with other disturbances, as well as when the test was repeated at intervals during recovery from the head injury. We administered and scored the Rorschach test according to the instructions of Klopfer and Kelley[11] except that the "testing of the limits" was omitted in order to avoid invalidation of repetitive tests during the course of recovery. From the Rorschach scores obtained in this standardized manner certain derivatives were obtained, as described elsewhere in detail.[12] These were the ratings for "instability," which were high for psychoneurotic patients, and of "disability," which were high for patients with cerebral disease. In table 3 are reviewed the levels of these ratings for various groups of subjects, including those with post-traumatic headache, psychoneurotic patients, with tumor of or damage to the brain and two groups of controls, one of superior intelligence, with a probable mean intelligence quotient of over 120, and one of low average intelligence, with a probable mean intelligence quotient of about 90. The rating for "instability" was related not only to neurotic trends but to intelligence. It showed a negative correlation with the Binet word list (-0.51 ± 0.069), but it differentiated two groups of subjects differing in evidence of neurotic background, although the Binet word list does not do so. The "disability" rating showed a negative correlation (-0.46 ± 0.074) with the span of digits test,

7. Snedden, D. S.: A Study in Disguised Intelligence Tests, Contributions to Education no. 291, New York, Columbia University Teacher's College, 1927.

8. Sherman, I. C.: Study of Kraepelin's Continuous-Subtraction Test, J. Abnorm. Psychol. **18**:385-388 (Jan.-March) 1924.

9. Shipley, W. C., and Burlingame, C. C.: Convenient Self-Administering Scale for Measuring Intellectual Impairment in Psychotics, Am. J. Psychiat. **97**: 1313-1325 (May) 1941.

10. Abbott, W. D.; Due, F. O., and Nosik, W. A.: Subdural Hematoma and Effusion as Result of Blast Injuries, J. A. M. A. **121**:739-741 (March 6) 1943.

11. Klopfer, B., and Kelley, D. M.: The Rorschach Technique, Yonkers, N. Y., World Book Company, 1942, p. 448.

12. Ross, W. D., and Ross, S.: Some Rorschach Ratings of Clinical Value, Rorschach Research Exchange **8**:1 (Jan.) 1944.

but it revealed striking changes in relation to head injury which were not brought out by the span of digits test except in cases of extreme injury.

Table three shows that within three weeks of a severe injury to the head (3 to 4 on our scale) the "disability" rating was notably raised, while with only slight or moderate injury (0 to 2) it was not raised more than to the mean for neurotic patients while the "instability" rating was raised almost to the mean for such patients.

For patients more than one month after head injury the mean for the "disability" rating was about that for neurotic patients, regardless of the personality background, while the "instability" rating was significantly higher in the presence of evidence of a neurotic personality background (rating of 1 or 2 on our scale for previous personality background) than in its absence (rating of 0). No other item, such as severity of injury, time after injury, presence of an electroencephalographic abnormality or a clinical judgment of cerebral damage based on an accumulation of the various types of evidence, showed a significant correlation in the entire series of patients with cerebral injury more than one month after the accident.

When only the 43 patients among those without a background of previous unstable personality were studied for correlation with "instability" and "disability" ratings, certain suggestive trends were present, as indicated in table 4. These trends might prove significant in a larger number of patients: a slightly greater tendency to "disability" with severe injuries than with moderate injuries, and to its appearance within a period of two years than after two years, and an even greater tendency to "instability" with these two factors, i. e., the severity of the injury and the stage of recovery from injury. "Instability" ratings also tended to be higher with electroencephalographic and other evidence of changes in the brain, although the "disability" rating was about the same one month after injury in groups divided on the basis of this evidence.

The changes with recovery from injury, as determined by repeated examinations on the same patients, are shown in table 5. Patients with severe injuries showed high "disability" ratings, which manifested recovery with time, although for 1 patient (E. G.) there was no improvement in the "disability" ratings, and this patient continued to show clinical evidence of severe "organic" intellectual impairment. The "instability" ratings for the patients with severe injury, however, tended to rise with improvement in the "disability" ratings. The case of E. T. is particularly interesting. Of previously stable personality, this man had a severe contusion and during convalesence passed through a phase with symptoms of irritability and dizziness, recovering spontaneously in about nine months. In relation to this clinical situ-

TABLE 3.—*Incidence of Rorschach Ratings*

	Number of Patients	Instability		Disability	
		Mean	Standard Error	Mean	Standard Error
Psychoneurotic patients	50	17.9	± 1.53	8.4	± 1.3
Patients with "organic" disease of brain	24	8.75	± 2.42	25.1	± 2.69
Healthy persons of superior intelligence	50	3.6	± 0.66	1.1	± 0.75
Royal Canadian Army Medical Corps, 1940	50	14.1	± 1.20	7.6	± 1.06
Patients with post-traumatic headache					
Tests within 3 weeks of head injury:					
Slight or no evidence of cerebral damage (0-2)	10	16.4	± 2.85	7.3	± 2.92
Marked evidence of cerebral damage (3-4)	10	7.4	± 2.89	30.7	± 4.43
Test more than 1 month after head injury:					
Without neurotic background	43	12.1	± 1.24	9.2	± 1.61
With neurotic background	44	18.4	± 1.61	8.0	± 1.34
With seizures	11	18.7	± 3.95	16.8	± 3.51

TABLE 4.—*Nonsignificant Trends in Rorschach Ratings for Forty-Three Patients Without Previous Neurotic Background*

Rorschach Rating	Severity of Injury		Time After Injury		Electroencephalographic Abnormality		Evidence of Organic Changes		
	Moderate	Severe	Under 2 Yr.	Over 2 Yr.	Present	Absent	Absent	Moderate	Pronounced
Instability	12.1	17.0	14.3	11.0	14.0	16.1	13.5	15.0	19.0
Disability	9.6	11.8	10.1	8.3	8.7	8.1	10.0	8.6	9.3

ation, table 5 reveals a falling "disability" rating with a rising "instability" rating, which reached a neurotic level while he had these symptoms and returned to normal when he was "himself" again. There was no compensation factor in this case.

The patients with less severe injury showed less evidence of "disability" and more immediate elevation of the "instability" rating, followed by a subsequent fall in some cases, but not in others.

Although it is necessary to make interpolations in the far from complete evidence which we have obtained, it appears that head injuries are followed by features detectable by the Rorschach method, at first of a variable degree of "disability," similar to that associated with cerebral disease and then by a degree of "instability"

TABLE 5.—*Serial Rorschach Ratings Following Injury to the Head for Fifteen Patients* **

	Instability						Disability					
	Less Than 2 Wk.	2 Wk.-1 Mo.	1-6 Mo.	7 Mo.-1 Yr.	1-2 Yr.	Over 2 Yr.	Less Than 2 Wk.	2 Wk. 1 Mo.	1-6 Mo.	7 Mo. 1 Yr.	1-2 Yr.	Over 2 Yr.
After severe injury:												
E. T.	11	17	..	7	..	..	41	21	..	4	..	..
J. R.	0	..	..	20	..	..	13	..	..	0	..	..
A. D.	..	0	8	22	..	..	..	34	27	17	..	..
B. M.	6	13	..	..	..	..	42	— 9	..	..	..	..
E. G.	..	— 1	0	7	..	..	..	49	41	45	..	..
J. D.	..	..	11	..	24	..	..	..	31	..	17	..
J. T.	..	..	..	21	..	18	..	..	..	9	..	— 1
H. M.	..	..	..	33	36	..	..	..	..	10	6	..
F. P.	..	..	..	..	8	21	..	..	..	..	36	19
After minor to moderate injury												
L. K.	17	29	..	..	..	..	30	13	..	..	..	..
D. L.	26	17	15	..	..	..	7	10	2	..	..	..
D. W.	26	..	..	18	..	..	7	..	..	— 1	..	..
L. R.	24	..	..	39	..	..	2	..	..	— 1	..	..
R. R.	4	..	2	..	..	..	— 3	..	2	..	..	..
A. K.	..	..	..	..	8	10	..	..	..	..	2	2

* Periods indicated in the column heads are those at which the ratings were determined.

similar to that for psychoneurotic patients. The degree of "instability" may be related slightly to the severity of injury and the amount of cerebral damage, but it is much more closely related to the previous personality background. It appears that "the fault, dear Brutus, is not in our stars, but in ourselves!" Whether the increased "instability" is a direct result of cerebral trauma, or whether it has an indirect cause, in the personal problems encountered by the victim of such trauma, we do not think our evidence is sufficient to show. We hope that either Nature or the hand of war will yet provide us with a subject or subjects on whom a Rorschach test has been made *before* head injury, with an opportunity to repeat such tests at intervals during recovery!

The relation of the "instability" and "disability" scores to the type of headache will be considered in section 7.

6. SITUATIONAL FACTORS

Each patient was considered for the presence of factors in his life situation at the time of or after the accident which might have contributed to the presence or aggravation of symptoms by providing either a mental conflict producing tension or a secondary gain to be obtained as a result of symptoms. The distributions of the situational factors has been summarized in the last section of table 1 according to each type of headache and the types of control.

The columns in this section of the table may be summarized as follows: 1. Apparently, no contributing situational factors.

2. Workmen's Compensation payments. This column was divided into (*A*) factors not appearing to contribute to the symptoms, according to our understanding of the case, and (*B*) factors appearing to play a part, at least in the aggravation of complaints.

3. Military services, in the Army, Navy, Air Force or Merchant Marine, which presented a mixed situation of unpleasant duties and probable danger, and in the first three services, possible pension.

4. Draft. The patients were facing a call for military training.

5. Legal factors. A lawsuit concerning the accident was pending.

6. Individual problems. Although none of the general situations often associated with accidents were present, an individual problem might be related to the symptoms.

Many of the patients with situational factors related to compensation and service also had personal problems, but the cases were tabulated in the column only for the general factor. Two patients whose cases involved two of the gen-

eral factors, i. e., eligibility for compensation and draft into the army, were given halfscores in each of the pertinent columns.

The individual personal difficulties which were discovered involved a wide variety of human hopes and fears. One man had had a marriage proposal spurned by the one woman he had ever dared to take out, and this occurred just previous to his accident. One woman had pent-up resentment against her husband for taking in boarders, which involved more work for her. She acquired a headache which she blamed on a minor injury six months previously, for which her husband had been accidentally responsible. Another woman was worried about her relatives in Russia. These situations were probably only contributory to more basic personality difficulities, but a full understanding of each case appeared to involve the relation of the previous personality background, the effects of the accident and the life situation after the accident.

It will be seen in table 1 that individual problems alone rank third among the situational factors presented by the patients, with Workmen's Compensation payments coming first and service of a military nature second. The differences between the patients and the controls and between various groups of patients will be discussed in section 7. This situational factor must, of course, always be interpreted in relation to the whole physical and personal condition of the patient, but by treating it as a statistical item, sought in both patients and controls, we have hoped to avoid the subjective biases of interpretation.

7. SIGNIFICANT ASSOCIATIONS AND TRENDS

On the basis of table 1, comparisons can be made both of the patients and the control subjects and of the patients with various types of headache and the three types of control subjects.

After the data for the 8 control patients who had some headache but did not present themselves because of their symptoms are removed from the total body of data for patients presenting headaches of various types and from the data for patients with other symptoms, we may compare the results for the 68 patients seeking medical care for symptoms after head injury and our control group of 22 persons who returned for study at our request. The only types of evidence which differ significantly in these two main groups are the severity of injury and the situational factors.

The evidence for severity of injury is significantly greater ($X^2 = 3.95$) for the control subjects than for the patients. There are two reasons for this. Our control subjects included 3 with very severe injuries and persisting neurologic defects who were selected purposely to provide at least a small control group representing severe trauma. In addition, almost all our control patients were persons whose injury had been of sufficient severity to warrant admission to the Montreal Neurological Institute, whereas a fair proportion of the patients had not been hospitalized at the time of their injury. The only importance of this difference to us is that it illustrates that the control group did not, at any rate, suffer less trauma than did the patients.

It appears of greater importance, however, that, whereas the patients and the controls did not differ significantly with respect to previous personality background, electroencephalographic patterns or Rorschach ratings, they did differ strikingly in the incidence of discoverable situational factors. All the control subjects taken together had a low rating ($X^2 = 29.7$, which is highly significant) for these factors as compared with the patients who sought medical care. Although we aimed at an unselected control group with regard to these factors, this difference may be exaggerated because of the difficulty of getting controls to return who were in the armed forces or were busy in war industry. Again, there might be some distortion as a result of a greater willingness of the patients seeking help to discuss their problems. Nevertheless, this difference does suggest that the complaint of post-traumatic symptoms is often related to the patient's life situation, although he might have symptoms without complaining of them if his situation is satisfactory or if there is no gain in complaining. This is in accord with Brain's [5] report of a greater frequency of complaints among patients after industrial accidents than among those after road accidents.

TABLE 6.—*Age Distribution of Patients Admitted to the Montreal Neurological Institute from 1938 to 1942*

Complaint	Below 18 Yr.	18-50 Yr. of Age	Over 50 Yr.
Post-traumatic headache	6	91	10
Acute head injuries	257	374	126

In this connection it is of interest that post-traumatic headache and dizziness are rare complaints in children. In the few cases we have encountered the complaint was made by the mother, and the connection of the headache with the injury was extremely dubious. At Dr. Cone's suggestion, we sought evidence on the

frequency of post-traumatic headache as compared with the incidence of head injuries in children. This is presented in table 6 on the basis of the number of admissions of patients of different age groups to the Montreal Neurological Institute over a five year period. The ratio of the incidence of patients with post-traumatic headache to patients with head injury is significantly greater in the 18 to 50 year old group than in the group under 18 years of age ($X^2 = 43.4$).

Of the figures for patients with the different types of headache and for the controls, given in table 1, those which differ significantly from the rest of the series are in bold face, or are marked with a dagger when the significance is established by the combination of two or three groups. They may be summarized as follows: The ratings for unstable personality background are low for patients with localized headaches ($X^2 = 11.4$) and for controls without headache ($X^2 = 4.3$) and are high for patients with generalized and bizarre headaches taken together ($X^2 = 10.08$) and for patients with symptoms other than headache ($X^2 = 5.74$); the ratings for severity of injury were high for controls with organic defects ($X^2 = 7.4$); electroencephalographic ratings were high for patients with localized headaches ($X^2 = 10.02$) and for controls with organic defects ($X^2 = 8.89$), and the Rorschach "disability" ratings were high for patients with localized headache ($t = 2.5$) and low for patients with hemicranial headache ($t = 2.7$).

Apart from these statistically significant associations between each condition and each type of evidence, there are trends in the various types of evidence which corroborate each other and which suggest that in a greater number of cases further associations could be established.

The patients with localized headaches not only had low ratings for an unstable personality background and high electroencephalographic and Rorschach "disability" ratings, but tended to have high ratings for the severity of injury and the pneumoencephalographic changes and somewhat low ratings for Rorschach "instability" as compared with patients with other types of headache. This much of the evidence tends to substantiate Penfield's [13] criticism of "an unmerited diagnosis of traumatic neurosis" for patients with localized headache, which he described as indicating a meningeal mechanism. With respect to situational factors, however, the patients with localized headaches tended actually to rate higher than the other groups, if one includes all patients with compensation claims in order to avoid a bias in deciding when the compensation factor is relevant. This difference is not significant statistically, but it indicates that even the localized headache is not uninfluenced by the life situation of the sufferer. This fact may account for the inability of some authors [14] to separate patients with localized headache from patients with other types of headache which are influenced by personal factors. Patients with localized headache appear prior to injury to have been of a type superior to the patients in the other groups, to have suffered a more severe injury, to have more persisting electroencephalographic and pneumoencephalographic changes, to have lower Rorschach "instability" ratings but higher "disability" ratings, at least within several months of the injury, and to share with the other patients a relation of their symptoms to their personal setting.

The following case is representative of this group of patients: W. M., a 26 year old corporal in the Royal Canadian Air Force, had been admitted to the Montreal Neurological Institute in 1941 for a localized headache, which had appeared one and a half years after a severe injury to the head (graded 3). Spinal subarachnoid insufflation of air was done at that time, with the idea that the pain was due to subdural adhesions. When the patient was observed two years later, it was ascertained that he had been "cured" by the insufflation but that the headache had been related to a difficult personality situation, in which he worked long hours under an unsympathetic taskmaster. The patient, now a sergeant and doing well in his work, felt that a transfer to another station had been responsible for the improvement. He had had one or two bouts of headache since the treatment, in relation to fatigue and worry, which one would not expect if the basis of the headache had been stretching of adhesions by the insufflation. He did not give evidence of a previously unstable personality, and four years after the injury, when the effects of cerebral trauma were probably well resolved, the electroencephalogram was normal and he had low Rorschach ratings for both "instability" (+4) and "disability" (+1) and a high intelligence quotient (over 120). There was no pneumoencephalogram, as the air had been directed to the subarachnoid space in 1941, and air studies were not repeated in 1943, when he was free from symptoms.

In contrast to the patients with localized headaches, the patients with bizarre headache tended to rate highest with respect to a previously unstable personality and Rorschach "instability" and lowest with respect to severity of injury, electroencephalographic changes and Rorschach "disability" rating. Some of them showed large

13. Penfield, W.: Chronic Meningeal (Posttraumatic) Headache and Its Specific Treatment by Lumbar Air Insufflation: Encephalography, Surg., Gynec. & Obst. **45**:747-759 (Dec.) 1927.

14. (*a*) Symonds, C. P., and Lewis, A., in Discussion of Differential Diagnosis and Treatment of Post-Contusional States, Proc. Roy. Soc. Med. **35**:601-614 (July) 1942. (*b*) Denny-Brown.[1]

ventricles on pneumoencephalographic study in spite of a mild or doubtfully severe head injury, and it is questionable whether such a change may not indicate a constitutionally defective nervous system rather than the results of trauma. The condition of these patients has impressed us as resembling hysteria in that secondary gain, rather than anxiety, was important. The generalized and bilateral headaches tended to approach the bizarre type in many characteristics, while the occipital form tended to be intermediate between these and the localized headache and presented more definite evidence of anxiety and tension.

The patients with hemicranial headache seem to form a special group. They rated low with respect to severity of injury, electroencephalographic changes and Rorschach "instability" and intermediate ratings for most other characteristics. It is interesting, also, that, of the controls, those with headache tended to have higher ratings for unstable personality than those without headache, although they were not higher in Rorschach "instability" rating.

We believe, then, that these trends allow us to place some weight on the description of headache presented by the patient with respect to the relative importance of physical and psychologic factors in the production of the headache, although we agree with Denny-Brown [1] and with Symonds and Lewis [14a] that "physiogenic" and "psychogenic" features can never be completely dissociated in the living patient. Whatever the physiologic mechanism of the symptoms, an inverse relation between the previous stability of the personality and the evidence for physical and

TABLE 7.—*Data on Follow-Up Study of Treatment of Eighty-Two Patients with Post-Traumatic Symptoms*

	Total Number of Operations	Effect on Headache							Effect on Dizziness						
		No Change	Slight Improvement	Considerable Improvement	Cure	Improvement with Relapse to: Considerable Improvement	Slight Improvement	Same Condition as Before	No Change	Slight Improvement	Considerable Improvement	Cure	Improvement with Relapse to: Considerable Improvement	Slight Improvement	Same Condition as Before
Spinal insufflation	40	16	6	1	4	5	1	7	14	2	0	4	0	1	1
Cranial insufflation	35	3	5	3	4	2	3	15	6	5	2	1	3	0	8
Encephalographic study	21	11	0	2	2	3	0	3	7	0	1	2	1	0	1
Direct surgical measures	8	0	0	3	3	0	0	2	1	1	0	2	0	0	1
Totals	104	31	11	9	13	10	4	27	28	8	3	9	4	1	11

"disability" ratings. In view of the fact that these were patients seen on an average longer after injury than the patients with localized headache, their headache might represent a later and milder form of the latter. On the other hand, all 3 patients showed moderate evidence of previously unstable personality background, in spite of fairly high intelligence quotients and good Rorschach ratings. It may be that their headache was akin to migraine, with which there is often an unstable background, together with a superior intellectual level. These patients, in common with patients with migraine, were also helped by the administration of ergotamine tartrate. At any rate, this type of headache is not frequent and tends to undergo spontaneous improvement.

The patients without headache but with other symptoms, such as dizziness, fainting spells, irritability, "nervousness" and fatigue, had high ratings for previously unstable personality and situational factors was striking. Most of the cases could be understood in terms of the constitution of the patient prior to the injury, its modification by the injury and the situation with which the person was faced. Hence, although further light on the physiologic mechanisms of post-traumatic symptoms would be useful in the management of the patients, we cannot get away from the need to consider each patient as a whole.

8. REEVALUATION OF THE SUBDURAL INSUFFLATION TREATMENT

A summary of our follow-up study on 82 patients who had previously been treated in this institute for post-traumatic headache or dizziness is presented in table 7. It includes the data on patients for whom we have been able to obtain follow-up information from six months to eight years after a spinal or a cranial subdural insufflation, as compared with the evidence for a control series for whom only a diagnostic en-

cephalogram was made, and with the results for a few patients on whom direct operative procedures were carried out. The data show the results of 104 procedures carried out on 82 patients, several patients having been submitted to more than one procedure. Of these 82 patients, 34 had returned because of failure of the operation to relieve their symptoms or because of a later relapse. Another 46 patients to whom we sent follow-up letters, and who were subjected to one of the three indirect procedures, including control encephalographic studies, did not reply. The results for these patients might have increased the percentage of cures, lowered as it was by the patients selected because of failure.

The fact remains, however, that we are able to report few cases in which the patient was cured or considerably improved several months to a few years after the procedure. The patients with spinal insufflation, like those with the ordinary pneumoencephalographic study, most frequently showed no essential change which could be attributed to the procedure, while a few who were subjected to this procedure shared with the patients with cranial insufflation a tendency to improvement, with later relapse. This relapse, with both procedures, usually occurred three to eleven months after the operation, the free interval accounting for the previous reports of short term cures. When relief of headache, both permanent and temporary, is considered for the patients with spinal insufflation, the figure (17 out of 40 patients, or 42.5 per cent) corresponds more closely with the results reported by Penfield and Norcross [15] (56 per cent) than does the long term result.

Of the patients subjected to spinal insufflation, shown in table 7, one who was cured of the headache was the corporal in the Royal Canadian Air Force the other factors in whose case have already been discussed; another patient returned because of the persistence of dizziness, but claimed that he had had no headache for two weeks *prior* to the insufflation.

At the suggestion of Prof. Wilder Penfield, and with the help of Prof. Allen O. Whipple, of the department of surgery of the Presbyterian Hospital in New York, we obtained reports on 3 of the 7 patients on whom the method of spinal insufflation was first tried by Penfield.[18] One (case 4 in Penfield's series) had been completely free from symptoms since the insufflation, which was done two months after a minor injury with no unconsciousness, a condition which in our experience suggests a tendency to spontaneous improvement if personality instability and situational factors are not severe. One patient (case 7) had had frontal headache only a few times a year, and the other (case 2), who had had a return of dizziness, as reported by Penfield, later had both headaches and dizziness until about five years ago, when they gradually cleared up spontaneously, leaving him with diminished vision and weakness of the left side, due no doubt to the cerebral damage from the original injury.

Of our patients cured of headache by cranial insufflation, one had been followed only three months and another only four and one-half months, when he died of pulmonary tuberculosis. In the case of a third patient the previous personality background and dramatic recovery of the sense of smell following the operation suggested that the result may have been psychologic. We have no further information on the fourth patient except that he still had lapses of memory, although his headache was cured.

In the original account of this procedure by Penfield and Norcross,[15] 6 patients were reported on, at a few months to a year and a half after the operation, as follows: patient 1 was better, but not cured; patient 2 was greatly improved, but not cured; patient 3 was improved; patient 4 although cured of headaches still had occasional dizziness; patient 5 had been relieved of the typical headache, and the dizziness was improved but still present, and patient 6, according to a recent report from the family physician, was free from all complaints. A subsequent review of these patients' charts provided the following more recent information: patient 1, after eight months, was still improved; patient 2, after four years, was still improved but had headaches about once a week although these were decreasing in frequency and were brought on chiefly by wet weather or by drinking beer; patient 3, after three years, remained a problem because of mental disturbance and still claimed to have severe headaches; patient 4 wrote to Dr. Penfield three years after operation saying that the headache had returned; patient 5 had a different kind of headache, and a year and a half after the operation the condition was diagnosed as psychoneurosis, and patient 6 returned not long after the receipt of the letter from his physician, claiming that he had had only temporary relief.

Even when we included the patients who presented other factors that might have been responsible for the cure and the patients with only short follow-up observations, the proportion of patients whose headache was cured or

15. Penfield, W., and Norcross, N. C.: Subdural Traction and Post-traumatic Headache, Arch. Neurol. & Psychiat. **36**:75-94 (July) 1936.

considerably improved or had recurred, with considerable improvement nevertheless, was no better in the group subjected to spinal insufflation (10 out of 40) or in the group with cranial insufflation (9 out of 35) than in the group with pneumoencephalographic therapy (7 out of 21), a treatment not considered specific for post-traumatic headache. This suggests that factors other than the insufflation may determine the result. There may have been patients, especially among those from whom we did not receive follow-up replies, whose headache was specifically affected by the insufflation treatment. A simultaneous psychiatric study of such patients would be required to establish the responsibility for cure, and we are unable to report any cases in which a long term cure was effected and other factors were definitely ruled out.

We tried to correlate the failures of spinal insufflation with failure to get air into the subdural space. However, in 2 of the patients who were cured the air entered the ventricles and not the subdural space, and in a considerable number of the patients who were not relieved of headache the air was in the subdural space. All the procedures were carried out by competent members of the attending or the house staff, presumably in accordance with instructions designed to insure a successful result. The fact that in a number of cases the air did not reach the subdural space may mean that this result is even more difficult to achieve than has formerly been thought.

The results of direct operation in the few carefully selected patients subjected to such a procedure seemed somewhat more specific, although this method of treatment of course carried more risk than the insufflation. In 2 patients the operation involved the cutting of adhesions when traction produced at operation, with the use of a local anesthetic, had reproduced the patient's pain. One of these patients, the cause of whose headache may not have been traumatic, has been under observation ten months, with no return of headache. The other patient showed temporary improvement but had a relapse within six months. One patient was rendered free of symptoms by the severance of adhesions and the repair of a defect in the skull with a tantalum plate. One patient had considerable improvement in her headache after the drainage of a subdural effusion, by Dr. Elvidge, although she still had symptoms, which were considered to have a neurotic basis. Two patients were relieved of pain by section of the fifth nerve, but in 1 of these, who had a cranial insufflation, an epithelioma of the face developed in the area of denervation, and he never returned to work, although he is still living and free from pain. The other patient continued to have dizziness, and even some headaches.

It appears that if a persisting anatomic abnormality is the factor which is holding up the patient's recovery, it should be dealt with, but that one must consider the patient as a whole and his setting before one can be sure that such is the case.

9. VASCULAR RESPONSES TO POSTURE

The responses of pulse and blood pressure to changes in posture have not been included in table 1, as these showed no apparent association with the type or to the presence or absence of headache, although there appeared to be some relation to dizziness. At first, we tried a tilting table test, as described by Weiss,[16] in which the subject was gradually elevated over a period of twenty minutes from a horizontal position to one of 80 degrees from the horizontal. In a study of 26 patients, however, we found that the changes in pulse rate and blood pressure were substantially the same with a simple measurement of these factors after the subject had been lying down for five minutes and then had stood for two minutes. On the basis of previous studies on university students in average physical condition, we concluded that a fall in blood pressure of more than 10 mm. and an increase in pulse rate of more than 20 beats a minute are unusual. Of 89 patients in our series, 28 showed such deviations. Of these 28 patients, 20 had dizziness, but 23 others complained of dizziness without these changes.

These vascular changes appeared to be related to the previous activity of the patient, whether he was in bed or was carrying on at his work, rather than to the severity or the type of the injury, apart from the confinement to bed induced by the injury. A few patients presented normal changes in pulse rate on admission to the hospital, and the deviation increased after they had been in bed for five days, after a pneumoencephalographic test. An increase was observed in a few neurotic patients without head injury and in an occasional healthy person of asthenic build. Such postural changes in pulse have been reported as present in cases of the effort syndrome,[17] and there may be a question whether the alteration is related to sedentary activity or to certain emotional features often present. At any rate, it does not seem related

16. Weiss, S.: Syncope and Related Syndromes, in Christian, H. A.: Oxford Medicine, New York, Oxford University Press, 1943, vol. 2, chap. 8 A, p. 250.

17. Lewis, T.: The Soldier's Heart and the Effort Syndrome, ed. 2, London, Shaw & Sons, Ltd., 1940.

specifically to head injury, and certainly not to the headache after such injury, although it is possibly related to the dizziness in some patients.

Malone [18] ascribed the effect of neostigmine to an assumed vascular instability. We conducted a trial of neostigmine [19] with 6 patients who had dizziness and gave a positive response to the postural change. We could see no improvement as compared with the effect obtained with placebo pills given these patients at another time. It appears that the confidence of the physician in such a measure must be ruled out before the pharmacologic action can be given the credit. It is of interest that with large doses of neostigmine (3 cc. of a solution containing 0.0005 Gm. of neostigmine methylsulfate per cubic centimeter, covered with 1/100 grain [0.6 mg.] of atropine sulfate, injected subcutaneously) in 2 of these patients we reduced the postural change in pulse rate—in 1 patient from 76 to 48 beats a minute and in the other from 38 to 32, but without alteration in their headache. In the second patient the pulse rate fell 14 beats after he returned to work. By the use of sodium nitrite we were able to increase the postural change in the pulse rate and the drop in blood pressure so that we could produce fainting in some patients, but we did not consider that this phenomenon was specific for post-traumatic conditions.

A routine of "tension exercises" has been prescribed for some time at this clinic by Dr. Cone, on the basis of the value of improved muscular tone in reducing a tendency to vascular instability. Undoubtedly, many patients have been helped by these exercises, perhaps in part psychologically, but we have seen the treatment fail with some patients because they still had unsolved personal difficulties. We have seen other patients helped as much by engaging in exercises for relaxation of large muscles, such as walking, dancing, gymnastics and swimming.

10. MECHANISMS, WITH IMPLICATIONS FOR MANAGEMENT

The factors which appear to be related to the production of symptoms in the patients we have studied are shown graphically in the diagram. Every injury to the head involves trauma, both physical and psychic. The physical effects are exerted on the meninges and the brain. Meningeal contusion and meningeal adhesions may be directly responsible for a localized type of headache mediated largely through the nerve supply to the dura from the fifth nerve.[20] This headache may clear up spontaneously if no other factors are in operation. It may persist for anatomic reasons and require surgical intervention, but the persistence may have an emotional basis, which is discussed later. Meningeal adhesions might also be responsible for dizziness, although such a cause is not well established. Localized headache may be produced by cerebral contusion, although we do not know what the anatomic pathways would be in such a case. Many of the localized headaches appeared to wax and wane with the presence and absence of fatigue and emotional stress, a feature suggesting a relation to cerebral function rather than to meningeal adhesions, although changes in intracranial circulation might provide the connecting link. Other effects on the brain include intellec-

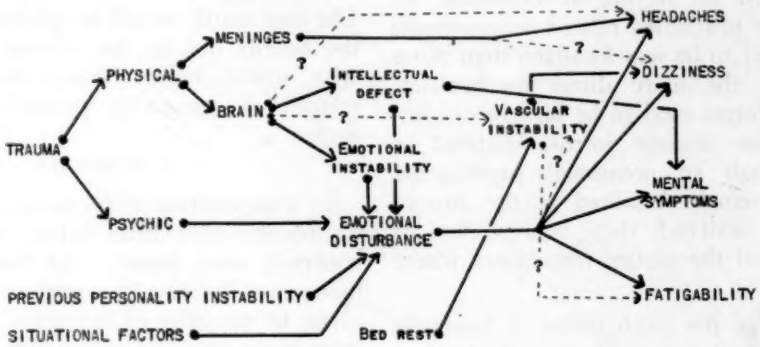

Diagram showing relation of various factors to the production of symptoms in patients with post-traumatic headache.

18. Malone, J. Y.: Head Injuries: New Treatment for Postconcussional Headaches and Dizziness; Preliminary Report, J. A. M. A. **119**:861-864 (July 11) 1942.

19. The neostigmine used in this study was donated by Hoffmann-LaRoche, Ltd., Montreal.

20. Penfield, W., and McNaughton, F. L.: Dural Headache and Innervation of the Dura Mater, Arch. Neurol. & Psychiat. **44**:43-75 (July) 1940.

tual impairment with severe injuries, and at least a temporary disturbance after less severe injuries, as well as emotional instability, as suggested by our studies with the Rorschach method. These features are linked with the psychic effects of the trauma, the previous personality of the patient and the situational factors to produce an emotional disturbance which may result in any of the symptoms of headache, dizziness, fatigability, irritability, lack of concentration and other mental disturbances so common in psychoneurotic patients. Anxiety or hysterical features may be present with these symptoms, according to the personality of the patient and other circumstances. These symptoms are probably produced through physiologic mechanisms, such as muscular tension and vascular instability, which are not yet fully understood. The emotional disturbance may also modify and prolong the reaction to a more direct mechanism, such as meningeal contusion or adhesions. The headaches related to emotional disturbances tend to be less localized than those associated with the more direct mechanisms. The hysterical forms tend to be bizarre or generalized, and the anxiety forms, bilateral or occipital, although an occasional psychogenic headache may remain localized at the site of injury in cases in which there was no loss of consciousness and the victim remembers where he was hit.

This concept of the mechanisms of headache indicates the importance of treating the patient as a whole, both for the prevention and for the management of post-traumatic symptoms. Neurosurgical precautions in the treatment of the acute injury are beyond the scope of our paper, but psychologic factors during convalescence may be mentioned. A reassuring attitude on the part of the neurosurgeon is desirable in order to allay the fears with regard to intellectual impairment and a possible threat to economic security, but it should not be an unwarranted optimism. If the patient is suffering a temporary impairment of mental efficiency and of emotional control, it is far better to recognize this and to give him an opportunity of getting back gradually to his previous functional level, rather than to lead him to think prematurely that he has made all the recovery he is going to make. Nor is there usually any need to expect a permanent disability, for in many patients the vis medicatrix naturae seems to persist, not only for months but for years after the injury, provided that the background is good and the environmental circumstances are not too adverse.

The treatment of the majority of patients with symptoms persisting for some time after the injury, for whom nothing can be done surgically, is essentially the management of psychosomatic conditions, with special consideration of any problems created by damage to the brain. Careful examination of the patient, careful consideration of the environmental factors and prolonged psychotherapy, all have a place. Palliative and sedative drug therapy will assist in the handling of the anxiety. Participation in relaxing recreation is also helpful. Above all, an "occupational therapy merging into therapeutic occupation," as Dr. Aubrey Lewis [14a] so aptly described it, should be employed. Pension and compensation boards would contribute to ultimate financial saving if they were to invest funds in establishments for the rehabilitation and reeducation of the injured, instead of continuing to pay out permanent partial disability pensions. The end result would be profitable, not only to the boards but to the victims of such injury, who would regain independent individuality instead of remaining precarious parasites on society.

SUMMARY

In a comparison of 68 patients with persisting complaints after head injury and 22 subjects observed after injury who had not presented themselves because of complaints, attention was given to the type of headache, the presence of items in the previous personality background suggesting instability, the severity of injury, the reports of electroencephalographic and pneumoencephalographic abnormalities, certain ratings of "instability" and "disability" as determined by the Rorschach method and situational factors which seemed relevant to the presentation of complaints.

The greatest difference between the patients and the controls lay in the presence of situational factors which might have produced or aggravated psychoneurotic symptoms. This was true for every type of headache. It was also evident that patients with localized headache showed less evidence of previous neurotic background and more evidence of cerebral damage from the injury. This evidence included electroencephalographic records and Rorschach "disability" ratings, with the ratings for severity of injury and pneumoencephalographic changes tending in the same direction, although less significantly. Except for a few patients with hemicranial headache, somewhat resembling migraine, patients with all other types of headache, especially those of bizarre and generalized distribution, showed previous personality instability similar to that of psycho-

neurotic patients and evidence of "instability" in the Rorschach test resembling that found for neurotic patients. The evidence for patients complaining of symptoms other than headache also tended to resemble that for neurotic patients.

A follow-up study of 82 patients with post-traumatic symptoms previously treated in this institute indicated that the long term results with spinal or with cranial subdural insufflation have been no better than the results following pneumoencephalographic treatment, and that for none of these patients could the influence of other factors be ruled out. The results in a few patients treated by a direct operative procedure suggest a limited application of such measures in carefully selected cases.

Studies of the postural vascular responses did not indicate any effect in patients with head injury which differed from that in psychoneurotic patients or in patients convalescing from other illness, nor have they given any lead to treatment other than to suggest the value of exercises for hardening and relaxation.

The concept of mechanisms presented here involves a relation between physical and psychologic factors and emphasizes the need of considering the person as a whole in the prevention and cure of post-traumatic symptoms.

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THE ELECTROENCEPHALOGRAM ASSOCIATED WITH EPILEPSY

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The present paper deals chiefly with the interpretation of the electroencephalogram when used as a diagnostic aid in the study of epileptic patients between seizures.

Hughlings Jackson [1] remarked:

> I submit that it is well also to consider that the warning in any paroxysm signifies the seat of a discharging lesion, that the warning is in that sense localizing.

Electroencephalography has shown that even preceding the warning there is frequently a localized abnormal electrical discharge from the brain, and in many cases this occurs almost constantly as a subclinical phenomenon. One therefore has an opportunity in a large number of instances of discovering the "seat" of origin of the potential changes, especially if the patient is studied between clinical seizures, when this may be localized, rather than at the height of a convulsion, when the electrical disturbance, like the clinical manifestations, may be generalized.

The problem of the electroencephalographic classification of the epilepsies has been approached from two standpoints. Gibbs, Davis and Lennox,[2] who pioneered in this field, showed that certain wave forms and patterns are frequently present during certain varieties of clinical seizures, and Gibbs, Gibbs and Lennox [3] proposed a classification based on these and other criteria. For instance, they stated:

> A fast rhythm spells grand mal, a slow rhythm psychomotor and an alternating slow and fast rhythm petit mal epilepsy.[3b]

Jasper and Kershman,[4] in attempting to use this classification in routine examinations of epileptic patients between seizures, reported that they could not accurately predict the form of the clinical attack from the wave forms and patterns seen in the electroencephalogram. They therefore undertook the problem anew. They carried out extensive localization studies on each patient, and as a result they concluded that localization of the abnormal discharge provided the most satisfactory basis for classification of the electroencephalographic records, with wave forms and patterns playing a secondary role in the detailed analysis of each case.

Evidence for and against the usefulness of these two classifications is found in the literature and will be outlined here. Walter,[5] by using the phase reversal technic introduced by Adrian and Yamagiwa,[6] was the first to demonstrate that an organic lesion of the brain, such as a tumor, reveals itself in the electroencephalogram by the presence of localized delta waves. Since then evidence has accumulated which shows that an epileptic cortical focus may likewise be localized (Golla, Graham and Walter [7]; Jasper and Hawke [8]; Gibbs, Gibbs and Lennox [3b]; Case [9]; Case and Bucy [10]; Jasper and Kershman [4]; Penfield and Erickson [11]; Gibbs, Merritt and Gibbs,[12]

From the Neurological and Neurosurgical Service of Lenox Hill Hospital.

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1. Jackson, J.: On Epilepsy and Epileptiform Convulsions, in Taylor, J.; Holmes, G., and Walshe, F. M. R.: Selected Writings of John Hughlings Jackson, London, Hodder & Stoughton, Ltd., 1931, vol. 1.

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3. (*a*) Gibbs, F. A.; Gibbs, E. L., and Lennox, W. G.: A Cerebral Dysrhythmia, Brain **60**:377-388, 1937; (*b*) Cerebral Dysrhythmias of Epilepsy, Arch. Neurol. & Psychiat. **39**:298-314 (Feb.) 1938.

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and others). The article by Casamajor and associates [13] should, perhaps, be added to this group, but these authors found that in a series of cases of unilateral seizures in children the abnormal rhythms, although tending to be more prominent in the hemisphere opposite the side of the convulsions, were always diffuse.

Concerning the interpretation of other aspects of the electroencephalogram in cases of epilepsy agreement is not so close. First, there is the difference of opinion expressed by Jasper and Kershman [4] and Gibbs and associates [3b] with respect to the value of wave patterns as diagnostic criteria, as previously mentioned. Rheinberger [14] favored the Jasper-Kershman point of view, and Hoefer [15] found much of value in the Gibbs-Lennox classification. In addition, Finley and Dynes,[16] in a recent review of the electroencephalograms of 369 patients with convulsive seizures, stated:

> Because of the great variety of patterns obtained in epileptics, and because many of these patterns resembled those found in other disorders, the use of clinical terminology in describing the brain wave patterns of epileptics is misleading.

These authors did not find the Jasper-Kershman or the Gibbs-Lennox classification satisfactory and stated the opinion that "the time has not yet arrived to make any formal classification of brain wave patterns." Finley,[17] in an analysis of 4,500 cases of neuropsychiatric disorders, also reported that he found rapid frequency cycles of the type described by Gibbs and associates as associated with grand mal to be more common with the involutional and manic-depressive psychoses and with neurosyphilis than with epilepsy.

The work to be reported is an attempt to interpret the electroencephalograms of 100 epileptic patients in the light of the conflicting observations and opinions just outlined. The first part of the study was devoted to an effort to classify the electroencephalographic records according to the localization of the abnormal discharges in the brain, as outlined by Jasper and Kershman,[4] and to tabulation of various wave forms and patterns seen, as described by Gibbs, Davis and Lennox [2] and Gibbs, Gibbs and Lennox.[3b] The results were correlated with the available clinical data, and certain conclusions were drawn.

With this information accessible concerning the localization of the abnormal waves and the types of clinical seizures of the individual patients, an endeavor was made to determine the significance of the slow wave patterns, so often seen, and described by Gibbs, Gibbs and Lennox,[3b] as of the "psychomotor" type. In addition, the possible significance of the so-called grand mal and petit mal wave patterns and of the frequency of the various waves is considered on the basis of these data. Finally, the nature of the neuron discharges which summate to form the high voltage waves in epilepsy is discussed.

It will be seen that the first part of the paper represents a repetition of the work of Jasper and Kershman.[4] This was necessary before the study reported in the second part of the article could be undertaken.

MATERIAL AND TECHNIC

One hundred and ninety electroencephalograms obtained during the past two and one-half years from 100 patients with a history of convulsive seizures were studied. The patients were all examined between seizures, but a number of them had clinical attacks during the recording. When this happened, a second examination was made later, so that tracings were obtained when the patient had been free of seizures (other than petit mal) for twenty-four hours before or after the taking of a record.

The neurologic and neurosurgical outpatient clinic at Lenox Hill Hospital, New York, provided the greatest source of the material. Patients in this group who showed focal cortical abnormalities were admitted to the neurologic-neurosurgical service for encephalographic study and more complete neurologic and electroencephalographic examination. In addition, a clinic was held at Lincoln Hospital for six months, and all patients with a clinical history of seizures suggesting a focal cortical onset were brought for electroencephalographic study through the outpatient department at Lenox Hill Hospital. A number of these patients were also admitted to the neurologic service for further investigation. A few patients with convulsive disorders were referred from Bellevue Hospital, and a group of patients were studied through permission of private physicians. A number of the last group were also referred for study because of a history of focal epilepsy.

The selection of patients with a history, and frequently neurologic signs, suggestive of a focal onset of their convulsions explains why the number showing

12. Gibbs, E. L.; Merritt, H. H., and Gibbs, F. A.: Electroencephalographic Foci Associated with Epilepsy, Arch. Neurol. & Psychiat. **49**:793-801 (June) 1943.

13. Casamajor, L.; Smith, J. R.; Constable, K., and Walter, C. W. P.: Electroencephalogram of Children with Focal Convulsive Seizures, Arch. Neurol. & Psychiat. **45**:834-847 (May) 1941.

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15. Hoefer, P. F. A., in discussion on Echlin, F. A.: The Electroencephalogram in Epilepsy, Arch. Neurol. & Psychiat. **49**:296-299 (Feb.) 1943.

16. Finley, K. H., and Dynes, J. B.: Electroencephalographic Studies in Epilepsy, Brain **65**:256-265, 1942.

17. Finley, K. H.: Potentials of Rapid Frequency in the Human Electroencephalogram, Arch. Neurol. & Psychiat. **49**:308-310 (Feb.) 1943.

localized cortical electrical abnormalities was so high (30 per cent).

A four channel, ink-writing electroencephalographic apparatus, built by Rahm, was used throughout. The apparatus was in one room and the patient in an adjoining one, which contained the input for the electrodes. This was connected to the amplifiers by means of a cable. Both rooms were electrically shielded, having been completely lead lined previously for use as x-ray rooms. They were shielded from one another by a grounded copper screen door. Nine electrodes were routinely used on each side of the head (fig. 1), and the placements were the same as those described by Jasper, Kershman and Elvidge.[18] Special electrodes were applied when indicated for localization. Localization studies were carried out on all patients, with the phase reversal technic, and bilateral recording was used during a considerable portion of the examination. Records were taken for one hour or more from most of the patients, the greater part of a morning or an afternoon being devoted to the study of each patient. The patient hyperventilated for four or five minutes during the study, and tracings were taken at intervals during this period.

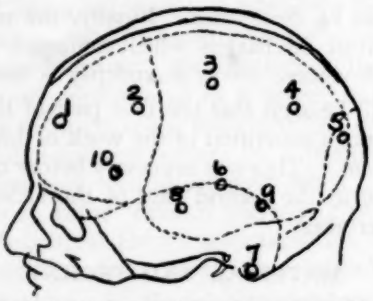

Fig. 1.—This diagram illustrates the position of the electrodes used in the present study. Placements *1* to *9* were employed (on both sides of the head) routinely in each case. The position of the electrodes is indicated (by numbers) to the left of the tracings in the following figures, and should be compared with figure 1 for proper interpretation.

Measurements used in the placement of electrodes were the same as those described by Jasper, Kershman and Elvidge.[18]

A sphenoid lead of the type used by Grinker and Serota[19] and by Jasper (cited by Penfield and Erickson[11]) to advantage in demonstrating the deep central origin of bilaterally synchronous potentials, was not employed in this study.

Miss Alice Wiltsek and Miss Share gave technical help in obtaining the records.

RESULTS

The most outstanding feature of the abnormal discharges recorded from epileptic patients was the recurring or paroxysmal appearance of high voltage waves, a phenomenon termed paroxysmal hypersynchrony by Jasper and Nichols.[20] Most, but by no means all, of these potential changes occurred at frequencies which may be regarded as abnormal, and therefore, in addition there was frequently dysrhythmia, as stressed by Gibbs, Gibbs and Lennox.[8]

It can be stated here that, although the discharges just mentioned constituted the outstanding feature of the records studied, similar abnormal waves (with the possible exception of the 3 per second wave and spike pattern) were seen in patients who did not have clinical epilepsy and it was therefore usually felt unwise to make a diagnosis of epilepsy from the electroencephalogram alone.

I. CLASSIFICATION OF ELECTROENCEPHALOGRAMS ACCORDING TO LOCALIZATION OF THE DISCHARGE

Under this heading are reported the results of an attempt to classify the electroencephalograms of 100 patients with a history of convulsions according to the Jasper-Kershman[4] classification. These authors reported that from the standpoint of localization three principal kinds of abnormal activity are observed in epileptic patients, namely: (1) localized (unilateral cortical), (2) bilaterally synchronous (from homologous areas of the right and the left hemisphere) and (3) diffuse.

In the present study it was found that the records taken from the 100 patients could be divided with a considerable degree of accuracy into the three chief types just mentioned. In addition, 20 per cent of the patients had what was regarded as a normal electroencephalogram.

Localized Unilateral Cortical Abnormality (30 per cent of patients)—In 30 of the patients studied between clinical seizures an abnormal discharge was recorded from a relatively restricted area over one cerebral hemisphere. The unilateral nature of the abnormalities in these epileptic patients immediately differentiated them from the bilateral changes in the electroencephalogram, to be described later. On figure 2 samples of subclinical unilateral discharges from 8 patients are shown.

It may be restated here that the patients examined were a selected group. When patients were referred from other hospitals, usually only those with a history or signs suggestive of focal epilepsy (12 patients) were chosen, and hence 30 per cent is not a true estimate of the number in an unselected group of epileptic patients who would have shown a localized abnormality in the

18. Jasper, H. H.; Kershman, J., and Elvidge, A.: Electroencephalographic Studies of Injury to the Head, Arch. Neurol. & Psychiat. **44**:328-350 (Aug.) 1940.

19. Grinker, R. R., and Serota, H. M.: Studies on Corticohypothalamic Relations in the Cat and Man, J. Neurophysiol. **1**:573-589, 1938.

20. Jasper, H. H., and Nichols, E. C.: Electrical Signs of Cortical Function in Epilepsy and Allied Disorders, Am. J. Psychiat. **94**:835-850, 1938.

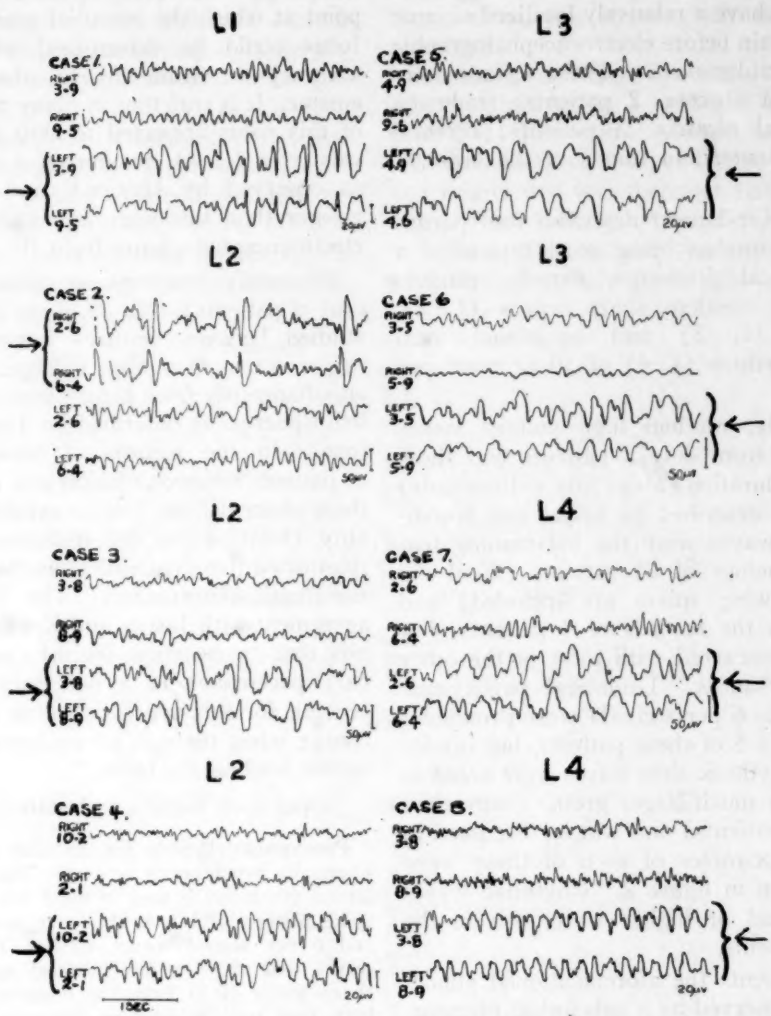

Fig. 2.—Samples of subclinical unilateral "focal" cortical abnormal discharges from 8 epileptic patients. For all patients tracings of the electrical activity were taken simultaneously from the two sides of the head, the upper two lines in each case representing the activity from the right side, and the lower two lines, that from homologous areas on the left side. The abnormal discharge is indicated by an arrow in the individual cases; this is confined to one side of the head, the left except in case 2.

The three common types of abnormal waves are illustrated in the tracings: *L 1,* spikes; *L 2,* sharp waves, and *L 3,* delta waves.

The distinguishing feature of the "focal" cortical abnormalities is their unilateral localized nature, and not the wave forms or patterns present, since these vary in form and frequency from moment to moment.

In cases 7 and 8 samples of rhythmic discharges (*L 4*) similar to those described as "psychomotor" by Gibbs, Gibbs and Lennox[8] were taken. In 3 of the other cases (1, 4 and 6) rhythmic slow waves patterns at 3 to 6 per second are also present, indicating the tendency of most discharges (in 50 per cent of cases with high voltage potentials in this study) at some time to take on a slow rhythmic form. Similar 3 to 6 per second waves are shown in 2 cases in figures 6 and 7.

Note the similarity between the unilateral 3 per second waves in case 7 and those in figure 3, case 5, and in figure 4, *3,* in which the waves are bilaterally synchronous. It will be seen, also, that the unilateral potentials in case 4 resemble the bilateral ones in figure 3, case 3.

The term "psychomotor" has been used in referring to all 3 to 6 per second rhythmic slow waves because of their essential similarity to one another and to the "psychomotor" pattern of Gibbs and associates,[8b] but it is not advocated that this clinical term be so used in the future.

That the potentials in certain instances presented at intervals a square top did not appear a sufficient criterion to warrant the separation of these patterns from the others into a so-called psychomotor group.

Electrode numbers are noted to the left of each tracing. Placement of electrodes is shown in figure 1.

electroencephalogram. Eight of the 30 patients were known to have a relatively localized organic lesion of the brain before electroencephalographic studies were undertaken. (These lesions were healed cerebral abscess, 2 patients; traumatic meningocerebral cicatrix, 3 patients; cerebral cicatrix, 2 patients, and tumor of the brain, 1 patient).

Jasper and Kershman [4] described four paroxysmal wave forms as being most typical of a localized cortical discharge, namely, random spikes (L 1), random sharp waves (L 2), delta waves (L 3) and occasional local paroxysmal rhythms (L 4) of 10 or more per second type.

In this study, random high voltage spikes were recorded from only 2 patients and these were of longer duration (about fifty milliseconds) than the spikes described by Jasper and Kershman.[4] Sharp waves were the outstanding feature in the tracings of 24 patients (26 if the 2 subjects showing spikes are included) and delta waves, in the records of 4 patients; but the latter also occurred with most of the other localized disturbances. Unilateral paroxysmal rhythms (at 3 to 6 per second) were prominent in the records of 5 of these patients, but briefer discharges of rhythmic slow waves were noted in the records of a much larger group (more than 50 per cent of patients) and will be discussed in detail later. Examples of each of these wave forms are shown in figure 2. Rhythmic waves at 14 per second or higher were seen in the records of 3 patients.

In all 30 patients the aforementioned abnormalities were observed as a subclinical phenomenon (without having the patient hyperventilate) in the absence of seizures for twenty-four hours before or after the tracing was taken. In fact, these localized discharges occurred as an almost constant phenomenon in over 65 per cent of the (30) patients with the unilaterally abnormal electroencephalogram. The subjects who had seizures during the taking of a record were studied again later. Hyperventilation, as described in the section on "Material and Technic," was used with all patients, and in over 50 per cent it caused some increase in the localized activity, either during or shortly after it was completed.

It was previously stated that the abnormal discharge was recorded from a relatively restricted area. This area, with no exception, was at least 4 cm. in diameter and varied of course with the intensity of the activity. When this increased, which occurred at some time in over 75 per cent of subjects, some effect was noted on the waves in the opposite hemisphere. By means of the phase reversal technic, the approximate point at which the potential gradients came to a focus could be determined with considerable accuracy to within an area about 4 cm. in diameter. It is true that in many cases the margins of this focus appeared to shift at certain intervals from 1 to 3 cm. during a single recording, as observed by Hoefer [15] and others. Shifts greater than this were not noted in subsequent electroencephalograms from the same patient.

Bilaterally Synchronous Abnormality (35 per cent of patients).—In 35 of the epileptic patients studied between seizures intermittent or paroxysmal bursts of high voltage waves appeared simultaneously from homologous areas in the two hemispheres, as described by Jasper and Kershman. In the records of about 40 per cent of patients hyperventilation was necessary before these abnormalities became evident. The bilaterality (location) of the disturbance clearly distinguished these patients from those who showed unilateral abnormality. The findings are in agreement with Jasper and Kershman's [4] hypothesis that "a common central (midline subcortical) pacemaker is a necessary hypothesis to account for this synchronization of bilateral discharge when there is no evidence for one hemisphere leading the other."

Jasper and Kershman [4] stated:

> Paroxysmal rhythms are the most common forms of bilaterally synchronous activity. The most perfect bilateral synchrony is seen in the 3 per second wave and spike pattern (B3 and 1), 3 per second waves (B3) and 6 per second waves (B 4). The only random waves that show a fair bilateral synchrony are the sharp waves (B 2) from the temporal lobes, but even here they tend to become rhythmic (frequencies between 3 and 6 per second) when showing bilateral synchrony.

These observations held true in the present study. An analysis of the records, based on their classification, follows. Examples of the four important paroxysmal rhythms seen in the records of patients with bilaterally synchronous abnormality are shown in figure 3.

Three per Second Wave and Spike (B 3 and 1) Activity: Seven records showing 3 per second wave and spike pattern were obtained from different patients between clinical seizures (figs. 3 and 4).

Sharp Waves (B 2) Activity: Bilateral sharp waves from the region of the temporal lobes were obtained from 14 patients between clinical attacks.

Three per Second (B 3) Activity: Abnormality of this type was present as the outstanding feature in the records of 13 patients (fig. 3). In 2 patients the 3 per second wave activity was

replaced by a 3 per second wave and spike pattern during a clinical seizure, and their records were therefore classified as having a bilaterally synchronous 3 per second wave and spike pattern.

In patients who showed bilaterally synchronous 3 per second wave or 3 per second wave and spike activity it was usually noted that between bursts of the high voltage waves the normal potentials were disturbed by fluctuations in amplitude and frequency. At times the alpha rhythm was replaced by waves fluctuating between 3 and 9 per second, as shown in figure 4. In some cases the abnormality was slight and was confined to the changes shown in figure 4, *2*, even during hyperventilation, and only on subsequent recording were sudden bursts of typical large voltage 3 per second wave and spike activity seen. In view of these observations, it appears that a relatively quiescent, deep, apparently midline focus may produce only a slight effect (diffuse disorganization) on the cortical rhythms, and hence the record might be mistaken for, and classified as, one showing diffuse nonlocalized activity.

Rhythmic Six per Second (B 4) Activity: Rhythmic waves at 3 to 6 per second will be described later. One patient had only intermittent discharges of 6 per second smooth waves, and this record was therefore not included with any of the foregoing types.

Diffuse Nonlocalized Activity (15 per cent of patients).—Only 3 patients showed diffuse nonlocalized abnormal activity similar to that described by Jasper and Kershman.[4] Two other patients showing such changes were excluded from the present series, as they were known to have gross diffuse lesions of the brain, and the presence of delta waves made interpretation of the records uncertain. It should be added that no children under 10 years of age were included in this study because of the difficulty in interpretation. Patients who had just recovered from an epileptic seizure and those in stupor or with

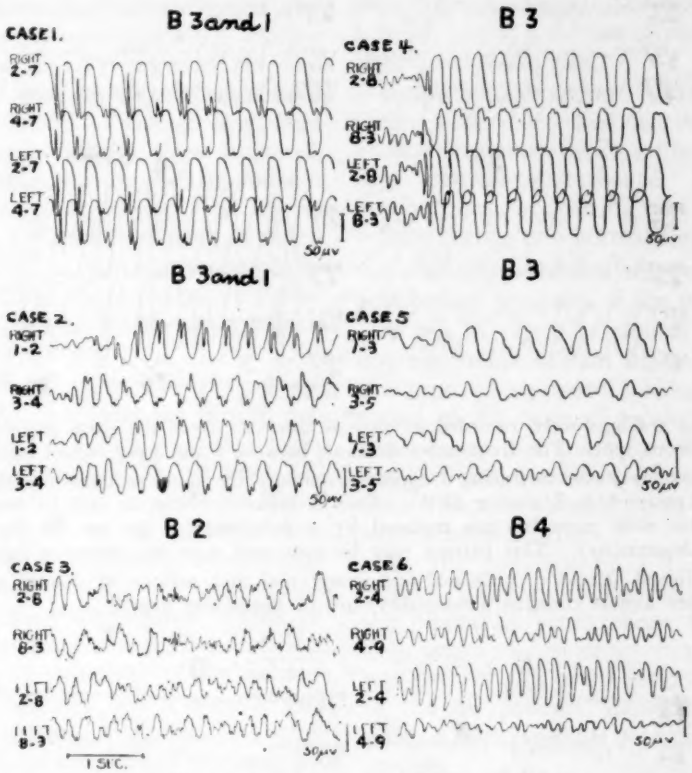

Fig. 3.—Discharges of the bilaterally synchronous type taken from 6 patients. *B 3* and *1* is an example of 3 per second wave and spike activity; *B 2*, of sharp waves; *B 3*, of 3 per second waves; and *B 4*, of 6 per second waves. The abnormal potentials in these cases appear simultaneously from the two hemispheres, in contrast to the potentials shown in figure 2, which were confined to one side of the head. In cases 3 and 5 the potentials are similar to those described as "psychomotor" by Gibbs and associates.[3b] Note the similarity between the wave patterns in these cases (3 and 5) and the rhythmic activity shown in figure 2. Tracings were taken simultaneously from the two sides of the head. The upper two lines in each case are from the right side of the head, and the lower two lines, from homologous areas on the left side. Electrode numbers are indicated to the left of each tracing, and their placements are shown in figure 1 (see text).

pronounced increased intracranial pressure fell into the same category.

However, 15 per cent of the patients did show a slight diffuse abnormality in the electroencephalogram (fig. 5). The characteristics of this disturbance were a moderate variation in the voltage and frequency of the alpha rhythm and the presence of scattered random delta waves of low voltage. These abnormalities frequently occurred in bilaterally synchronous fashion. Discharges of rapid low amplitude sharp waves at 14 to 25 per second were occasionally present. A dividing line could not always be drawn between records of this type and an electroencephalogram judged to be normal. The 3 patients with more conspicuous diffuse abnormality were included in the 15 per cent.

Normal Electroencephalograms.—Twenty per cent of the epileptic patients studied had normal records (fig. 5).

CORRELATION OF ELECTROENCEPHALOGRAPHIC AND CLINICAL DATA.

Under this heading the clinical data concerning the variety or pattern of the epileptic seizure are correlated with the location of the discharge in the brain and with the wave pattern seen in the records of certain patients. This represents a repetition of Jasper and Kershman's [4] work.

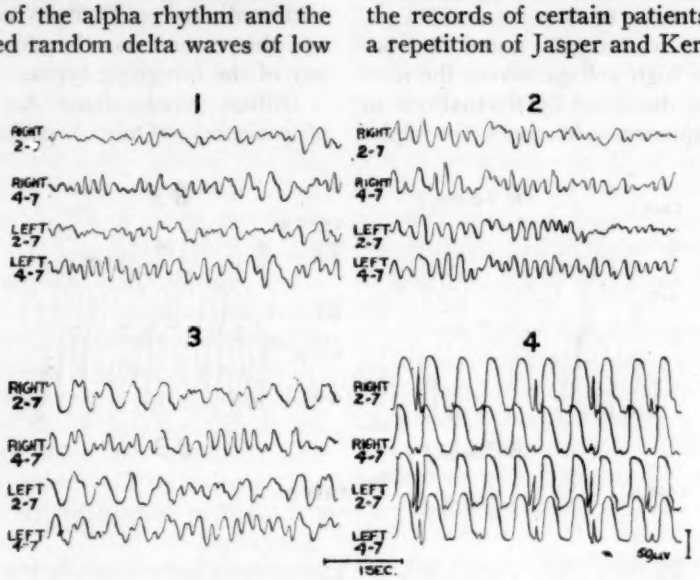

Fig. 4.—These four tracings were recorded at various intervals from the same patient. The activity is of the bilaterally synchronous type. The frequencies fluctuate between 9 and 3 per second (see text for explanation).

The slow (3 per second) waves in tracing *3* appear essentially the same as those described as "psychomotor" by Gibbs, Gibbs and Lennox (fig. 3, tracing 18 [3b]). This is interesting, for, as will be seen, in tracing *4*, taken thirty seconds later, the slow potentials are replaced by a subclinical 3 per second wave and spike pattern (so-called petit mal abnormality). This pattern may be compared with the waves in figure 2, case 7.

This patient had clinical attacks of petit mal and grand mal and periods of mental dulness. His electroencephalogram presented almost constant abnormality, but of fluctuating degree.

Fig. 5.—Record *A* shows a slight diffuse abnormality, and *B* is a normal electroencephalogram (see text for explanation).

Classification by Clinical Seizure Pattern.—In classification of the various forms of clinical seizures an endeavor has been made to employ the terms "psychomotor," "psychic variant or equivalent" and "amnesia" in the sense in which Gibbs, Gibbs and Lennox [3b] used them. However, it is probable that many of the attacks placed in this category would not be regarded as "psychomotor" by Gibbs and associates because of their minor nature. In part II of the paper the clinical and electroencephalographic aspects of such episodes will be discussed in more detail.

Clinical seizures were considered to be of the focal cortical or of the petit mal type, according to the criteria outlined by Penfield and Erickson.[11] Petit mal attacks are presumably due to a disturbance originating deep near the midline of the brain and as a rule are apparently represented in the electroencephalogram by bilaterally synchronous abnormal waves when an active discharge is present. Minor attacks commencing in a localized area of the cortex, such as might result from a discharge in the region of the left temporal lobe, may give rise to a transitory sensory aphasia (disorientation, confusion and transitory disturbance of consciousness). Such a minor attack is not usually regarded in this paper as a petit mal seizure but, rather, is considered as a "psychomotor" (psychic equivalent or variant or amnesia attack) (see figure 3, case 3, for a record taken during such an attack). Undoubtedly, brief attacks of this type are at times mistaken for petit mal. However, when they are accompanied in the electroencephalogram by a focal cortical abnormality, their true nature becomes apparent, if the interpretation used in this paper is correct.

Focal Cortical Seizures: Twenty-eight patients were regarded as having attacks the clinical pattern of which suggested a focal cortical origin. The electroencephalograms of 75 per cent (21 patients) showed a relatively localized unilateral abnormality, and those of 2 patients with clinically focal seizures, bilaterally synchronous sharp waves. The records of 5 patients showed a slight diffuse disturbance. This information, based on localization studies, indicated that the majority (75 per cent) of the patients with focal clinical seizures had a specific type of electroencephalogram. Classification, however, was impossible on the basis of the form and pattern of the waves alone, for individual subjects presented a wide variation and intermittent fluctuation in these characteristics when the activity increased. Patients with focal seizures of this kind frequently had a history of psychomotor attacks, "psychic equivalent or variant" states, episodes of "amnesia" or grand mal seizures; in addition their attacks had a progressive march, and at times a distinctive aura. In fact, some patients, at one time or another, had all these forms of clinical seizures, and hence localization of the abnormality was the method of choice in classification of their disorder.

Petit Mal Attacks: Nineteen patients had what were clinically called petit mal attacks. On the basis of studies between clinical seizures, the electroencephalogram of 1 of these patients indicated a definite localized cortical disturbance (and, as previously mentioned, the condition should therefore probably not be classified as petit mal); 12 patients presented activity of the bilaterally synchronous type; 4, slight diffuse abnormality, and 2, normal electroencephalograms. Only 3 of these patients had petit mal without a history of grand mal seizures as well. These 3 patients showed bilaterally synchronous subclinical activity of the 3 per second type.

"Psychomotor" Attacks, "Psychic Variant or Equivalent" states and "Amnesia": Thirty-four patients gave a history of attacks of this type. Twelve had focal cortical abnormality in the electroencephalogram; 15 bilaterally synchronous activity, and 7, a diffuse disturbance or a normal electroencephalogram.

Grand Mal Attacks: There were 86 patients with a history of grand mal seizures. As a subclinical phenomenon, 24 subjects showed a localized cortical disturbance, 30, a bilaterally synchronous abnormality and 32, either a slight diffuse change or a normal electroencephalogram.

Comment.—The following observations appear justified on the basis of the preceding data:

When the form of clinical seizure is known, one may make certain limited predictions as to the type of electroencephalogram that will be found on the basis of localization studies. For instance, in the majority of patients (75 per cent) clinical focal cortical seizures were associated with a localized cortical abnormality in the electroencephalogram.

In the presence of a history of petit mal attacks, the abnormality was almost always of the bilaterally synchronous type (suggesting a deep origin) when an active subclinical discharge was present (12 of 19 patients with petit mal, 6 of the 19 patients showing relatively no abnormal high voltage potentials at the time of study).

In the case of psychomotor attacks or grand mal clinical seizures no predictions could be made concerning the type of electroencephalogram (from the standpoint of localization) that would be associated, for all types of electroencephalographic patterns, unilateral localized cortical, bilaterally synchronous, diffuse and normal, were well represented.

RELATION BETWEEN LOCATION OF DISCHARGE AND TYPE OF CLINICAL SEIZURE

Clinical Seizures Associated with a Localized Unilateral Cortical Type of Abnormality (30 patients).—Seventy per cent (21) of the patients who had electroencephalographic patterns of this type gave a clinical history of focal epilepsy. For 53 per cent (16) of these patients a pathologic cortical lesion was demonstrated by air studies, operation or autopsy, and in each patient

the lesion was at or near the site indicated by the electroencephalogram. Twenty-four (80 per cent) of the 30 patients included in the series gave a history of grand mal attacks; 12 patients had "psychomotor" attacks, "psychic variant or equivalent" states or episodes of "amnesia," and 1 patient, petit mal. The average age of the patients in this group was 25 at the time of study.

Clinical Seizures Associated with Bilaterally Synchronous Activity (35 patients).—Thirty (85.7 per cent) of the patients with electroencephalographic abnormalities of this type had grand mal seizures, and 28 (80 per cent) had such attacks without any clinical pattern to suggest an origin in a focal area of the cortex. Two patients (5.7 per cent) gave a history of attacks the focal clinical pattern of which suggested an origin in the temporal lobe, and these patients had bilaterally synchronous sharp wave (B_2) activity. Twelve (34.2 per cent) of the patients had petit mal, and 15 (42.8 per cent) "psychomotor" attacks, "psychic variant or equivalent" states or episodes of "amnesia."

Bilaterally Synchronous Three per Second Wave and Spike (B 3 and 1) Activity: Seven patients had this electroencephalographic pattern. All of them had grand mal attacks without localizing features, and 5 gave a history of petit mal attacks as well. One patient also had "psychomotor" episodes.

Bilaterally Synchronous Three per Second (B 3) Waves: Thirteen patients showed electroencephalographic patterns of this type. Nine of the patients had generalized grand mal seizures, 5, petit mal seizures and 6, "psychomotor" attacks, "psychic variant or equivalent" states or episodes of "amnesia." Three patients gave a history of petit mal attacks only, without any grand mal seizures. Patients showing the B 3 and 1 or B 3 type of abnormality had an average age at the onset of their attacks of 12 years, and their average age at the time of study was 18 years.

Bilaterally Synchronous Sharp Waves (B 2): Fourteen patients had electroencephalographic patterns of this type. Thirteen patients gave a history of major seizures. Eight patients had "psychomotor" attacks, "psychic variant or equivalent" states or spells of "amnesia," in addition to grand mal seizures, and 2 patients had petit mal, as well as grand mal, seizures. In 2 patients the pattern of the attacks was focal (symptoms suggesting an origin in the temporal lobe).

Rhythmic Three to Six per Second (B 4) Waves: This pattern will be considered later.

Clinical Seizures Associated with Slight Diffuse Abnormality or Normal Electroencephalograms.—Thirty-five patients (35 per cent) had such records. As previously mentioned, it was difficult to draw a dividing line between many of the records showing a slight diffuse abnormality and normal electroencephalograms. This was equally true in clinical analysis. The patients have therefore been analyzed as a group.

Thirty-two (91.4 per cent) of the patients had grand mal epilepsy; 7 patients had "psychomotor" attacks, "psychic variant or equivalent" states or episodes of "amnesia"; 6 had petit mal, and 5 had focal clinical seizures. Twenty-seven patients gave a history of grand mal without any clinical localizing features.

The average age at the onset of attacks in this group was 26, and the average age at the time of study was 37.

Comment.—From the form of the electroencephalogram (classified according to localization of the disturbance) guarded predictions can be made concerning the type of clinical seizure with which it may be expected to be associated.

1. When a localized cortical disturbance was present, one could surmise for a high percentage of patients (70 per cent in this study) that focal clinical seizures would be associated. However, 40 per cent of these same patients had "psychomotor" attacks (psychic equivalent or variant or amnesia states), and 80 per cent had grand mal episodes in addition.

2. When bilaterally synchronous waves were the outstanding feature in the electroencephalogram, grand mal seizures, without localizing features, were the rule (80 per cent of patients), and petit mal attacks were common (34.2 per cent). Of the patients with a history of grand mal, 93.3 per cent (28 of 30 patients) had such seizures, without any clinical evidence to suggest that they originated in a localized area of the cortex. These observations are in keeping with the hypothesis that the origin of the electrical abnormality lies deep in the brain in these cases. "Psychomotor" seizures were also frequent (42.8 per cent) in this group.

3. Patients with normal electroencephalograms or a slight diffuse electrical disturbance (relatively quiescent abnormal activity) had seizures essentially similar to those in the preceding group except that clinical attacks of the focal cortical type were a little more frequent (14.3 per cent).

II. WAVE PATTERNS ASSOCIATED WITH EPILEPSY

Gibbs, Davis and Lennox [2] and Gibbs, Gibbs and Lennox [3b] observed that certain wave patterns were present during particular types of

clinical attacks. An attempt to determine the significance of these observations, especially as concerns their "psychomotor" form of activity, is presented in this part of the paper. To begin with, the electroencephalographic aspects will be considered, and later these will be correlated with the clinical data.

"PSYCHOMOTOR" TYPE OF ELECTRICAL ACTIVITY

Gibbs and associates [3b] described their "psychomotor" activity as consisting of

> . . . cortical action potentials which are of high voltage and slow frequency—from 3 to 6 per second. There are series of regular, square-topped waves with the normal rhythm of from 8 to 10 per second still present on the crest. . . . At another period of the seizure the waves, though still of high voltage, are more irregular and without the crenated crests.

Similar activity occurring as a subclinical phenomenon is also referred to as "psychomotor." were seen. This was also found to be true by Finley and Dynes.[16] The waves referred to as square topped (figs. 2 to 4, 6 and 7) usually alternated with smooth or sharp forms, even in tracings from patients with the most severe "psychomotor" clinical manifestations.

Electrically one such discharge seemed essentially the same as another. In view of this nonspecificity in the changes in slow potential, all high voltage 3 to 6 per second rhythmic wave discharges were classed as "psychomotor" even though some of them were of short duration and occurred only occasionally in a record. This was done purely to facilitate analysis, and it is believed that in the future it would be preferable to drop the term "psychomotor" in referring to electroencephalographic patterns.

In the analysis to follow, slow wave patterns will be considered under three headings, according to their localization in the brain.

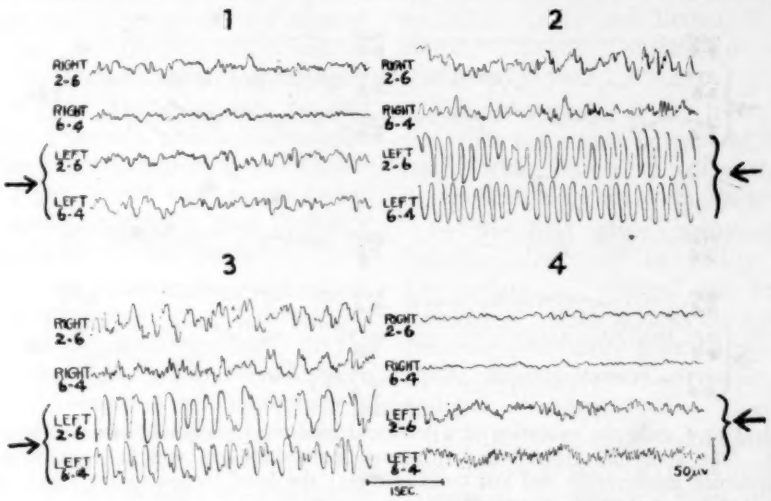

Fig. 6.—Unilateral localized cortical abnormality, illustrating the evolution of the discharge during a clinical attack. Tracings were taken simultaneously from homologous areas on the two sides of the head. The record was taken six minutes before (*1*), during (*2* and *3*) and four minutes after (*4*) a "psychomotor" clinical seizure, in which the patient showed mental confusion and uncontrolled movements. The lesion in this case was an area of cortical atrophy in the left occipitotemporal region, demonstrated by air encephalogram. The patient had frequent visual auras and occasional grand mal episodes. When his localized discharge increased in intensity, as shown in the present tracings, it involved the left occipitotemporoparietal region and gave rise to mirror activity in the opposite hemisphere. The same slow pattern, when less intense, was associated with a visual aura alone on one occasion. The waves are smooth topped during the height of the "psychomotor" clinical and electrical disturbance and later show considerable variation in form, as do most of the 3 to 6 per second potentials in other figures in this paper (see text).

In the records studied in the present series, 3 to 6 per second rhythms were a common feature (see percentages cited later). It was not possible, however, to say which of these slow rhythmic disturbances should be classed as "psychomotor." No truly square-topped waves, of the "textbook" form of Gibbs and Gibbs,[21]

21. Gibbs, F. A., and Gibbs, E. L.: Atlas of Electroencephalography, Cambridge, Mass., Lew A. Cummings Co., 1941, p. 16.

Localized Unilateral Cortical Disturbances.—In studying the discharges of localized cortical type, it was found that the abnormal waves occurred at random, as described by Jasper and Kershman.[18] However, in over 50 per cent of the 30 patients showing this form of abnormality it was noted that the random potentials were replaced at some time during the recording by rhythmic trains of high voltage slow waves, with a frequency of 3 to 6 per second. In the records

of 5 patients the slow rhythmic waves occurred frequently and for many seconds at a time. In the other records these discharges were infrequent and of shorter duration. However, it is believed that all these discharges were essentially the same as those described as "psychomotor" by Gibbs, Gibbs and Lennox [8b] (figs. 2, 6 and 7), although the waves were not always square topped. Such slow rhythms were especially likely to occur when the activity increased in intensity (voltage) and spread to involve a somewhat larger area of cortex (so long as it did not become generalized, as in a grand mal attack). In approximately 50 per cent of the patients presenting this phenomenon (localized unilateral rhythmic discharges of 3 to 6 per second waves), the discharge was brought about by hyperventilation. In the others it occurred spontaneously. Examples of these slow wave patterns are shown in figures 2 (cases 1, 4, 6, 7 and 8), 6 and 7.

The rhythmic waves just described were all noted subclinically in patients who did not have a clinical seizure for at least twenty-four hours before or after the tracing had been taken.

In 1 patient similar 3 to 6 per second waves were recorded during a clinical attack. This subject, as a subclinical phenomenon, presented a localized unilateral cortical discharge of random waves (from the left occipitotemporal region), which at intervals became of higher voltage and rhythmic, with a frequency of 3 to 6 per second. At the onset of the clinical attack the random waves were replaced by higher voltage smooth potentials at 3 to 7 per second, as shown in figure 6. During the period of high voltage discharge, and for a short period thereafter, the patient became confused and out of contact and attempted to pull the electrodes off his head. He had what could be termed a "psychomotor" attack. It is interesting that his clinical manifestations were unlike his usual seizures, which as a rule consisted of a visual aura, at times followed by a grand mal convulsion. In the episode described the temporal lobe, as well as the occipitoparietal region, was involved, and some mirror activity appeared in the opposite hemisphere.

Another patient had a subclinical localized cortical type of disturbance in the left temporal region, made up of random waves which at intervals, as in the other case, became rhythmic and of higher voltage, with a frequency of 3 to 6 per second. This patient had a sudden change in his cortical potentials during the taking of the tracing, as shown in figure 7. As will be seen, the random unilateral waves were replaced by rapid ones, which became intermittently rhythmic at 3 to 6 per second, and three seconds later high voltage fast waves spread over the entire head. Clinically the patient exhibited a fixed stare just preceding the appearance of the high voltage generalized fast waves; his head turned to the right, and he then had a grand mal seizure.

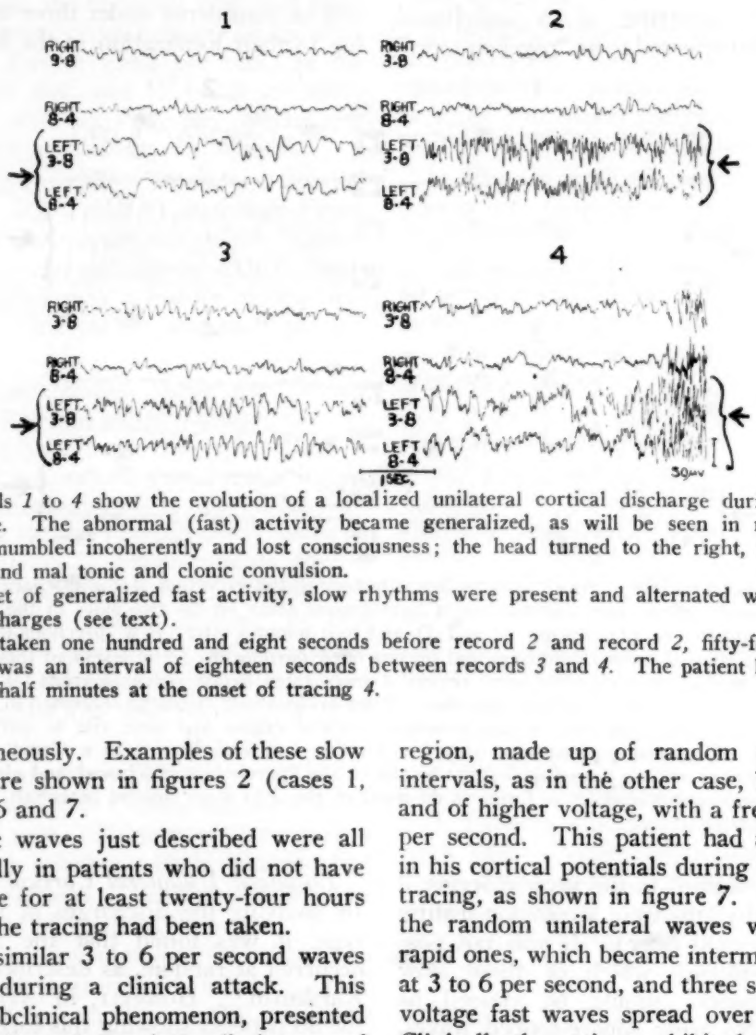

Fig. 7.—Records *1* to *4* show the evolution of a localized unilateral cortical discharge during the onset of a grand mal seizure. The abnormal (fast) activity became generalized, as will be seen in record *4*. At this point the patient mumbled incoherently and lost consciousness; the head turned to the right, and a few seconds later he had a grand mal tonic and clonic convulsion.

Before the onset of generalized fast activity, slow rhythms were present and alternated with fast (high frequency) wave discharges (see text).

Record *1* was taken one hundred and eight seconds before record *2* and record *2*, fifty-four seconds before record *3*. There was an interval of eighteen seconds between records *3* and *4*. The patient had hyperventilated for three and one-half minutes at the onset of tracing *4*.

The clinical significance of these observations will be considered later.

Bilaterally Synchronous Cortical Activity.—In over 65 per cent of patients (9) presenting bilaterally synchronous potentials of the sharp wave (B 2) type, particularly over the temporal lobes, rhythmic trains of high voltage waves at 3 to 6 per second occurred at some time during the taking of the electroencephalogram. The patterns formed were similar to those called "psychomotor" by Gibbs, Gibbs and Lennox.[3b] The tracing in figure 3, case 3, illustrates such activity.

Three per Second Wave and Three per Second Wave and Spike Pattern: In the records of patients with bilaterally synchronous 3 per second wave and spike patterns it is not uncommon (2 of 7 patients) to see trains of 3 per second smooth or square-topped waves as a subclinical phenomenon (fig. 4, *3*). There does not appear to be any fundamental difference between these 3 per second waves (especially the square-topped forms) and the rhythms described as "psychomotor" by Gibbs, Gibbs and Lennox (fig. 3, tracing 18 [3b]). The same may be said of the 3 per second smooth and square-topped waves seen in the electroencephalograms classified as showing 3 per second wave activity (fig. 3, case 5). All such disturbances have therefore been classified as being of "psychomotor" type.

Comment.—From the observations just stated, it appears that "psychomotor" (3 to 6 per second wave) patterns are common (present in the records of over 50 per cent of patients with high voltage epileptic disturbances) and may occur in a localized unilateral cortical discharge, in one arising deep in the brain in the neighborhood of the temporal lobes or in one apparently originating near the midline. Such patterns, therefore, have no specific location of origin in the brain. Localization of these potential changes, then, would be one method of classifying them.

POSSIBLE CLINICAL SIGNIFICANCE OF CERTAIN WAVE PATTERNS IN THE ELECTRO-ENCEPHALOGRAM

Exception has been taken to the use of clinical terms, such as "psychomotor," petit mal and grand mal, to describe certain patterns in the electroencephalogram.[22] This criticism is based on the fact that similar electrical disturbances are seen as a subclinical phenomenon in patients who never had clinical seizures of the type implied by the name used to describe the changes in the electroencephalogram.

22. Finley.[17] Finley and Dynes.[16] Jasper and Kershman.[4]

The important observation of Gibbs and associates [3b] remains true, however, namely, that certain electroencephalographic patterns frequently occur during particular types of clinical seizures. There must be some explanation for these phenomena. A hypothesis is advanced in the "Summary" which may explain their association.

In the next paragraph is considered the question of what clinical manifestations may be included under the term "psychomotor." An attempt is then made to determine what relation exists between "psychomotor" subclinical discharges and "psychomotor" clinical seizures. The significance of the observations will be left for consideration in the final discussion, in which the possible significance of electroencephalographic patterns associated with petit mal and with grand mal seizures will also be analyzed.

"Psychomotor" Clinical Seizures.—The clinical seizures associated with this form of electrical activity, it will be recalled, were described by Gibbs, Gibbs and Lennox [3b] as of "psychomotor," "psychic variant or equivalent" or "amnesia" type.

Now it appears, if my interpretation is correct, that not a specific type of epileptic seizure, but a wide variety of epileptic manifestations, is represented by these clinical terms.

In the first place, many episodes termed "psychomotor" are in reality expressions of automatism, as pointed out by Penfield and Erickson.[11] Such attacks are apparently usually (but not necessarily always) release phenomena, either during (ictal) or after (postictal) an epileptic discharge.

In addition to episodes of automatism, it seems that certain psychic seizures usually regarded as arising in the temporal lobes, attacks of sensory aphasia and, in fact, any seizure (apart from typical petit mal) accompanied by mental confusion (transitory or otherwise) that is not immediately associated with a clonic or generalized convulsion must be included with the "psychic variant or equivalent" or the "amnesia" type. Such an interpretation has been used in naming and classifying seizures in this paper, and as a result all such clinical episodes are referred to at times as "psychomotor," for the sake of convenience.

CORRELATION OF CLINICAL MANIFESTATIONS AND LOCATION OF "PSYCHOMOTOR" DISTURBANCES

"Psychomotor" Clinical Seizures and Localized Unilateral Cortical Discharges.—Of the 30 patients with cortical discharges of this type in the electroencephalogram, 12 gave a history of "psychomotor" attacks, "psychic equivalent or

variant" states or episodes of "amnesia." Nine of these patients had subclinical 3 to 6 per second rhythmic wave patterns in their electroencephalograms, and 3 of them did not show such changes. It will be remembered that at least 15 (50 per cent) of the 30 patients with localized cortical activity had 3 to 6 per second discharges (figs. 2, 6 and 7). Therefore 6 patients had these electrical disturbances without any history of psychomotor or variant clinical seizures. In the electroencephalograms of 5 patients (as previously mentioned) the psychomotor electrical pattern was a prominent feature in contradistinction to those showing only brief trains of rhythmic 3 to 6 per second waves. Four of these 5 patients gave a history of clinical symptoms of a more pronounced "psychomotor" nature than any of the other patients with similar episodes.

For the 9 epileptic patients who had both electrical and clinical "psychomotor" patterns, the location of the abnormal discharges was as follows: left temporal region, 4 patients; left frontal lobe, 1 patient, and left parietal or occipital lobe in the region of the left angular gyrus, 4 patients. Of the patients with rhythmic 3 to 6 per second waves, but without clinical "psychomotor" symptoms, the disturbance was localized as follows: left temporal region, 1 patient; right frontal region, 1 patient; left parietal region, 2 patients; right parietal region, 1 patient, and left occipital region, 1 patient.

Psychomotor Seizures and Bilaterally Synchronous Sharp Waves (B 2 Activity).—As previously stated, over 65 per cent (9) of the 14 patients with electroencephalograms showing bilaterally synchronous sharp waves over the temporal lobes had rhythmic wave discharges at a rate of 3 to 6 per second at some time during the recording. Eight of the 14 patients (with bilateral sharp wave activity) had a history of "psychomotor" clinical attacks, and 5 of these 8 patients had 3 to 6 per second rhythmic potential changes as well. Four patients with 3 to 6 per second waves had no history of "psychomotor" clinical attacks, and the slow rhythmic patterns were somewhat less prominent in these persons.

Psychomotor Seizures and Three per Second Wave (B 3) and Wave and Spike (B 1 and 3) Activity.—Of the 7 patients with subclinical 3 per second wave and spike patterns, only 1 gave a history of "psychomotor" clinical seizures. Six patients, however, of the 13 patients from whom bilateral 3 per second waves were obtained had such clinical episodes. It is debatable whether 3 per second smooth waves should be regarded as "psychomotor" in type. It is true, however, that the smooth waves frequently changed from moment to moment to the square-topped form typical of the "psychomotor" patterns, as shown by Gibbs and associates (fig. 3, tracing 18 [3b]) and illustrated here (fig. 3, case 5). The "psychomotor" symptoms presented by most of these subjects could be described as of the behavior problem type or as periods of mental dulness.

In the group under discussion, square-topped 3 per second waves, as shown in figure 3, case 5 and figure 4, *3*, were as common in patients with "psychomotor" clinical seizures as in those with no history of them (see "Comment" and "Summary" for further consideration of the significance of these observations.)

POSSIBLE SIGNIFICANCE OF FREQUENCY IN ELECTROENCEPHALOGRAPHIC DISTURBANCES

Partly because slow rhythmic waves accompany psychomotor seizures and fast potentials grand mal attacks, it has been suggested that "high frequencies produce overactivity and slow frequencies underactivity." [8b] If this observation is correct, it would have a fundamental bearing on the interpretation of the electroencephalogram and therefore deserves careful consideration.

It is well known that when the abnormal activity in any case of epilepsy becomes generalized and results in a grand mal attack, the onset of the convulsion is frequently accompanied by high voltage fast waves (as described by Gibbs, Gibbs and Lennox [3b]). But at this stage of the episode the patient usually loses consciousness. In other words, the "high frequency" waves are accompanied by, or produce, underactivity (loss of consciousness), rather than overactivity, as suggested in the preceding paragraph.

Leading up to this stage in an epileptic seizure, it is not uncommon to see bursts of high voltage fast waves alternating with slow rhythmic potentials at 3 to 6 per second (fig. 7). To explain this phenomenon on the basis of the hypothesis under discussion, overactivity would have to alternate with underactivity every few seconds. No evidence to support this assumption has been found.

It will also be remembered that during the violent clonic phase of a convulsion slow rhythmic high voltage waves are a common feature of the electroencephalogram. This is also true during a "psychomotor" seizure, in which clouding of consciousness (underactivity) may be accompanied by rage or excessive activity in the motor sphere (as in the case illustrated in figure 6). It seems that in both these states it

might be argued that slow frequencies (waves) produce, or are associated with, overactivity, rather than underactivity.

Why high frequency potentials often accompany the onset of a grand mal seizure is not definitely established. It appears, however, that when the disturbance spreads rapidly, as in such a seizure, the cortical cells may behave in one of two ways: (*a*) They may fire off at relatively high frequencies and independently (at different rates), producing an asynchronous fast wave discharge (as shown in figures 7 and 8), or (*b*) they may discharge rhythmically and at high frequencies, apparently in a truly synchronous manner (comparable to that shown in figure 8). The rate of this discharge may be the same as the normal alpha or beta rhythm or may occur at a higher frequency. Other aspects of this hypothesis are discussed in more detail later.

After this stage in the evolution of the disturbance in a grand mal convulsion, large areas of the cortex (during the clonic phase of the attack) may show rhythmic periods of activity and rest, so that masses of cells fire simultaneously and then become quiescent; i. e., the rhythm (frequency of the waves) slows.

This tendency for a slow rhythm to appear seems characteristic of most abnormal electrical disturbances in the brain of epileptic patients when large masses of cells are involved in intense activity for any appreciable length of time. Such slow frequency waves seem to produce underactivity (in an epileptic attack), but only in the sense that physiologic function is disturbed when a large portion of the brain is taken over by excessive abnormal activity.

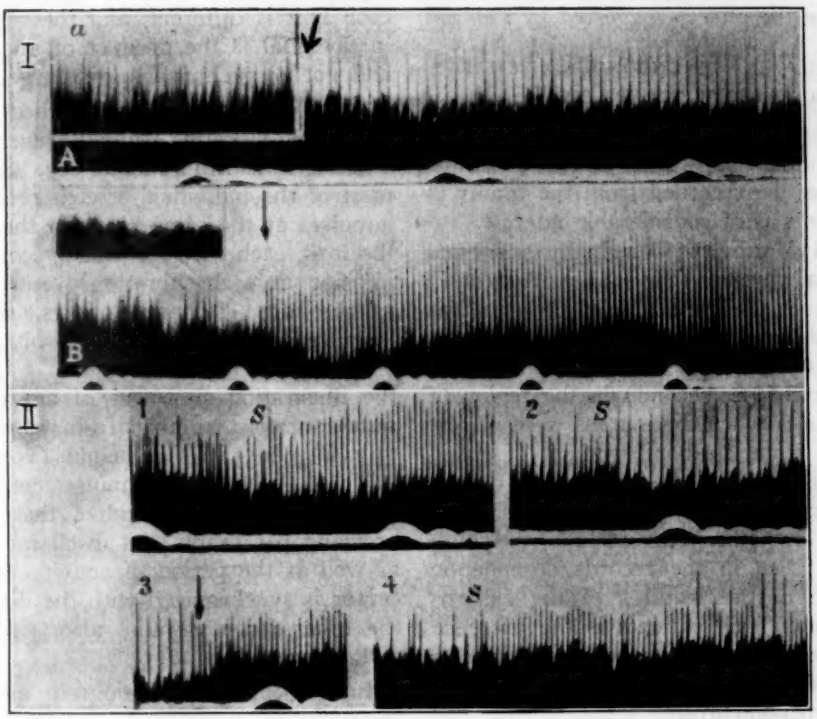

Fig. 8.—*I A*, insert *a*, and *I B*, to the left of the arrow, are examples of asynchronous discharges of impulses in nerve when steady tension is applied to the gastrocnemius muscle of the frog. The insert in *I B* shows the base line of the record before stretch was applied.

In *I A* and *B*, at the arrow in each case, are seen synchronous discharges of impulses from the stretch receptors when a tuning fork is applied to a string attached to the tendon of the gastrocnemius muscle.

In II, tracing *4* at *S* shows the type of waves produced by individual nerve impulses from a single receptor (in a frog's toe muscle). Tracing *1* and *2* at *S* show the type of waves resulting from the synchronous response of two receptors (see original article for further explanation). In each case the waves are of approximately the same duration regardless of the number of units involved and vary only in amplitude. This is in contrast to the waves in epilepsy, which usually become "slow" when large numbers of units discharge simultaneously (see text). The time marker indicates one-fifth second intervals (from Echlin, F., and Fessard, A.: J. Physiol. **93**:312-334, 1938).

In addition to the aforementioned observations, it should be noted that when one speaks of slow frequencies, one is referring to discharges of slow waves and not to the frequency of nerve impulses. As pointed out later, these slow potentials (in epileptic patients) are ap-

parently usually made up of high frequency discharges from individual neurons. The separate waves are due to the summation effect of a large number of these cells firing simultaneously, but apparently at high frequencies. The term "slow frequency discharge" in this sense is therefore misleading, for the individual neurons in such a "slow frequency" disturbance may be firing at as high rates as, or at higher rates than, those in a so-called fast wave discharge.

The observations just reported pertain to the abnormal high voltage electrical activity in the records of epileptic patients, and it is not disputed that in a nonepileptic discharge an increase or a decrease in frequency of the cortical potentials over that usually regarded as normal might produce the effects suggested by Gibbs.[23]

SYNCHRONOUS AND ASYNCHRONOUS DISCHARGES ASSOCIATED WITH EPILEPSY

The nature of the nerve impulse discharges which go to make up the various abnormal waves that may be recorded from the brains of epileptic patients is of considerable interest.

Adrian and Matthews[24] originally suggested that the alpha rhythm (8 to 13 per second) normally seen in the electroencephalogram was the result of the synchronous discharge of large numbers of cells (firing at their normal frequency of 8 to 13 per second) in the region of the occipital lobes when these areas were at rest or were freed from the bombardment of visual afferent impulses when the eyes were closed. Jasper and Nichols[20] and Jasper and Kershman[4] extended this hypothesis to include the high voltage waves seen in the records of epileptic patients. One of the features of the electroencephalogram of such patients is the presence of recurring outbursts of large voltage waves, a phenomenon described by these authors as paroxysmal hypersynchrony.

Before the waves in epilepsy are considered in detail, it is perhaps well to consider what is meant by a synchronous as opposed to an asynchronous discharge. The example to be given deals only with a fast wave disturbance, in which each receptor contributes only one impulse to each wave in the discharge.

Figure 8, *1*, shows the type of disturbance which results when the gastrocnemius muscle (in a frog) is stretched with a steady tension. In this case the many receptors in the muscle send out impulses at different frequencies, and the resulting discharge is a low voltage asynchronous one. The same type of asynchronous discharge of impulses occurs each time the muscle is stretched (unless the period of stretch is brief, as in the case cited in the next paragraph). This is true, for instance, if tension is applied to the muscle intermittently (rhythmically) at a rate of 3 per second (each stretch lasting approximately one-third second). In this case one might argue that the activity occurs synchronously in each receptor and that the periods are synchronized with the pacemaker (at 3 per second). However, although the periods of activity are approximately the same for all receptors, the rate of discharge of impulses from each unit is different, and the resulting change in potential is the product of an asynchronous, and not a synchronous, discharge.

Now, if the muscle is stretched intermittently by a rapid vibrating stimulus (such as that from a tuning fork with a rate of, say, 128 per second), most of the individual stretch receptors fire off impulses at the same rate (at the frequency of the fork, each receptor usually contributing one impulse at each vibration), and thus a truly synchronous discharge occurs, which in this instance is composed of high voltage fast waves occurring in rhythmic fashion. Each wave in the illustration shown is of approximately the same duration as that from a single impulse (fig. 8) and varies in height (voltage) according to the number of impulses contributing. In the case just given, unlike that cited in the previous paragraph, the discharge of impulses, as well as the period of activity, from each receptor is synchronized with the discharges from the other receptors and, also, with the tuning fork (pacemaker).

In the electroencephalogram associated with epilepsy, as mentioned previously, one sees occasional rhythmic series of high voltage fast waves. These are apparently true synchronous discharges, similar to the potential shown in figure 8. Individual high voltage spikes or multiple spikes are also apparently due to the synchronous discharge from a number of nerve cells and are therefore presumably the product of hypersynchrony.

Whether such an explanation, however, as implied by Jasper's[25] term "paroxysmal hypersynchrony," is sufficiently explanatory when applied to epileptic discharges in general appears to depend partly on the nature of the slow potential changes in the cortex. With the exception of "spikes" and certain fast waves, the

23. Gibbs, F. A.: Interpretation of the Electroencephalogram, J. Psychol. **4**:365-382, 1937.

24. (*a*) Adrian, E. D., and Matthews, B. H. C.: Interpretation of Potential Waves in Cortex, J. Physiol. **88**:440-471, 1934; (*b*) The Berger Rhythm: Potential Changes from the Occipital Lobes in Man, Brain **57**: 355-385, 1934.

25. Jasper and Kershman.[4] Jasper and Nichols.[20]

majority of the waves recorded from the cortex of epileptic patients are slow. There is some doubt concerning the origin of these slow waves. Gerard and Libet [26] apparently concluded that individual nerve cells may give rise to slow electrical potentials and that synchronous action of a group of such cells may produce a slow wave which is a close replica of that from a single cell except that it is of greater voltage. Adrian and Matthews [24a] pointed out that the activity of cortical neurons consists of a series of brief pulsations. The existence of slow waves in experimental epilepsy, they stated, implies asynchronous discharges in the different parts of the area which contributes to the potential gradient. From this point of view, the slow waves are summation effects of an excessive discharge of a large number of neurons firing asynchronously (at different frequencies), and the response of each unit bears no resemblance to them. Adrian and Matthews [24a] stated, however, that there must be a general agreement in the periods of rest and activity over considerable areas of cortex, although the neurons in these areas are not pulsating in phase. In epileptic human subjects the presence of smaller waves, which are often superimposed on the crest of the slow potentials, favors this theory of asynchronous firing of the individual units which go to make up the slow waves (figs. 2 and 4).

These observations are supported by more recent ones, in which Adrian and Moruzzi [27] (recording from single fibers in the pyramidal tracts of animals) demonstrated that the cortical waves in experimental epilepsy are usually summation effects from neurons firing impulses asynchronously, at frequencies sometimes as high as 500 to 1,000 per second.

If my interpretation of the observations of Adrian and his co-workers [28] is accurate, the term paroxysmal hypersynchrony (although of course correct) is not sufficiently explanatory of the physiologic mechanism concerned in the production of most of the recurring or paroxysmal high voltage slow potentials which can be recorded from the brains of epileptic patients.

It is true that the periods of abnormal activity in the various neurons (contributing to a slow potential) may occur synchronously with one another and with the so-called pacemaker (show periodic simultaneous intervals of activity and rest, i. e., hypersynchrony, as suggested by Jasper and associates [25]) but the resulting trains of nerve impulses in the separate axons apparently occur at different frequencies (as in the case of the activity of muscle receptors to relatively prolonged stretch), and the composite discharge of impulses (which presumably also contributes to the slow wave) is therefore asynchronous.

26. Gerard, R. W., and Libet, B.: On the Unison of Neurone Beats, in Ozoro de Almeida, A., and Ozorio de Almeida, M.: Livro de Homenagem, Rio de Janeiro, Brazil, 1939.

27. Adrian, E. D., and Moruzzi, S.: Impulses in Pyramidal Tract, J. Physiol. **97**:153-199, 1939.

28. Adrian and Matthews.[24] Adrian and Moruzzi.[27]

COMMENT

Although this study is based on a small number of patients (100), certain observations appear justified.

In only a little over 65 per cent of the patients with convulsive seizures studied were paroxysmal high voltage waves present in the tracings taken between clinical attacks. The remaining patients had either a normal electroencephalogram (20 per cent) or a slight diffuse abnormality, consisting chiefly of low voltage waves (15 per cent). With the exception, perhaps, of bilaterally synchronous 3 per second wave and spike discharges, the subclinical electrical abnormalities recorded could not be regarded as specific for epilepsy, and a diagnosis of epilepsy on the basis of the electroencephalogram alone was therefore rarely felt to be justified.

Probably the chief value of the present investigation is that it corroborates in large part the observations of Jasper and Kershman [4] that electroencephalograms taken from epileptic patients between seizures may be classified on the basis of localization studies into four groups, namely, (1) localized unilateral cortical abnormalities, (2) bilaterally synchronous abnormalities, (3) diffuse abnormalities and normal electroencephalograms.

The fact that convulsive states may be classified with considerable accuracy according to localization studies, in this manner, is of value from a clinical standpoint and, in addition, throws light on certain aspects of the physiologic mechanism of epilepsy. The observations reported are in keeping with the hypothesis that the discharge in most patients with epilepsy (over 65 per cent) may originate as a relatively focal or local disturbance and then, under certain circumstances, spread to other regions of the brain. The clinical manifestations resulting from such abnormal or excessive electrical activity appeared to depend largely on the function of the areas of the brain involved or influenced (and the intensity of the discharge). In other words, a correlation was found to exist between the patient's type of epileptic seizure and the location of his abnormal discharge (as

demonstrated by Jasper and Kershman,[4] Penfield and Erickson [11] and others).

The possible significance of the various wave forms and electrical patterns, the frequency of the waves seen in the electroencephalogram and certain physiologic mechanisms concerned in the production of these abnormal potentials will now be discussed briefly.

FOCAL OR LOCAL NATURE OF EPILEPTIC DISCHARGES

With regard to the evidence in favor of the localized origin of the different disturbances, one may consult the paper by Jasper and Kershman [4] and the recent book by Penfield and Erickson.[11]

There is no doubt that the attacks of a large number of epileptic subjects (30 per cent in the present selected series) have a localized unilateral cortical origin, as demonstrated by Golla, Graham and Walter [7]; Jasper and Hawke [8]; Case and Bucy [10]; Jasper and Kershman [4]; Penfield and Erickson [11]; Gibbs, Merritt and Gibbs,[12] and others. Evidence that the bilateral synchronous waves (35 per cent of patients in this study) originate in, or are controlled from, a relatively localized area, deep in the brain, either near the midline or in the neighborhood of one of the temporal lobes, has been discussed at length by Jasper and Kershman [4] and Penfield and Erickson,[11] and I shall not repeat it here, except to say that the electrical and clinical observations in this study are in keeping with the hypothesis expressed by these authors.

There is evidence (page 275) that the slight diffuse disturbance noted in 15 per cent of the patients (with abnormality relatively quiescent at the time of study) may occasionally be due to a feebly discharging area deep in the brain. Such an abnormality might, therefore, at times also be regarded as having a relatively local origin.

As already mentioned, the records of 20 per cent of the patients were considered normal. The epileptic discharges in these patients were, therefore, inactive at the time of study, and no conclusions can be drawn regarding the source of their seizures except from clinical criteria.

In summary, it may be stated that the observations are in keeping with the hypothesis that most epileptic discharges (over 65 per cent) apparently arise as relatively local or focal disturbances in the brain and that localization of these changes in potential by means of the electroencephalogram serves as one basis for classification of the epilepsies.

POSSIBLE SIGNIFICANCE OF WAVE FORMS AND PATTERNS IN THE ELECTROENCEPHALOGRAM

In the body of the paper, the observations concerned with the electrical and clinical patterns of so-called psychomotor, petit mal and grand mal were reported and commented on. These observations will be reviewed and discussed here.

Gibbs, Davis and Lennox [2] and Gibbs, Gibbs and Lennox [3b] were the first to report that 3 per second wave and spike patterns occurred during petit mal attacks; 3 to 6 per second, frequently square-topped, potentials, during "psychomotor" episodes, "psychic variant or equivalent" states or seizures of "amnesia," and high voltage fast waves, during episodes of grand mal.

It should be reemphasized here that the present report (as well as the much more extensive one of Jasper and Kershman [4]) is based largely on a study of electroencephalograms taken between seizures, and hence the results do not conflict with the important observations of Gibbs and associates,[29] during clinical attacks. The use of clinical terms like petit mal, psychomotor and grand mal to describe subclinical electrical patterns, however, is perhaps misleading, since such abnormalities occur in the electroencephalograms of patients who do not have attacks of the type implied, as demonstrated in this series and by Jasper and Kershman,[4] Finley and Dynes,[16] Finley [17] and others.

So-Called Psychomotor Clinical and Electrical Patterns.—Slow rhythmic high voltage waves at 3 to 6 per second were present at intervals in the records of over 50 per cent of the epileptic patients from whom high voltage abnormal waves were recorded. These slow rhythmic patterns were found in discharges arising from a unilateral localized area in the cortex, as well as in disturbances originating deep in the region of a temporal lobe or apparently deep near the midline of the brain (figs. 2 and 4).

Electrically there did not seem to be any fundamental difference between one of these slow rhythmic disturbances and another. The shape of the individual waves in most cases fluctuated from moment to moment from a sharp to a smooth or a square-topped form. This fluctuation in form appeared to depend chiefly on the changing intensity of the discharge, on its position in the brain and its relation to the recording elec-

29. Gibbs, Davis and Lennox.[2] Gibbs, Gibbs and Lennox.[3b]

trodes and on the degree of synchronization of the units participating. That the potentials in certain instances at intervals presented a square top did not in itself appear a sufficient criterion to warrant the separation of these records from the others, into a so-called psychomotor group. Actually, square-topped forms were almost as common in the records of patients with no history of psychomotor seizures as in those of patients with such seizures.

In the present study, all high voltage 3 to 6 per second rhythmic slow potentials have been classified as "psychomotor" disturbances to facilitate analysis. This seems justified because of the essential similarity of these abnormalities, but it is suggested that in the future it might be preferable to drop the term "psychomotor" in describing these electrical patterns.

Only about 55 per cent of patients with high voltage rhythmic 3 to 6 per second waves had what might be termed clinical attacks of "psychomotor," "psychic variant or equivalent" or "amnesia" type.[3b] All these subjects had other forms of clinical epileptic seizures, in addition to their "psychomotor" episodes. From this it is evident that 3 to 6 per second waves of the "psychomotor" type are common in a wide variety of epileptic discharges, that they have no specific site of origin in the brain and that they do not necessarily mean that a patient will have psychomotor clinical seizures. In other words, they are not specific for this type of attack. This is in keeping with the fact that they are seen during sleep and anesthesia and in animals, as well as in persons with no history of epilepsy, as reported by Finley and Dynes.[16]

The clinical manifestations which accompanied 3 to 6 per second waves varied widely in the cases presented in this paper. Even those episodes classified as psychomotor attacks, psychic equivalent or variant states or amnesia have not conformed to any specific pattern. In fact, it was felt necessary to include under this type all attacks (other than true petit mal) in which the patient showed a clouding of consciousness (transitory or otherwise), providing there was not an accompanying grand mal or jacksonian motor or sensory seizure. The reason for this variation in symptoms associated with 3 to 6 per second waves appeared to depend largely on the location and intensity of the electrical disturbance.

It is apparent that in an epileptic subject abnormal electrical activity of the slow rhythmic type would disturb physiologic function in the cells participating if its intensity was sufficiently great. If the area of brain involved in such a disturbance was large enough or was concerned with higher intellectual function (such as the left temporal lobe), it is easy to see how clouding of consciousness (psychomotor variant, psychic equivalent or variant or amnesia) might occur unless unconsciousness supervened. Similar activity confined to a more "silent" area, however, such as the right frontal or right temporal lobe, might give rise to few or no clinical manifestations unless the disturbance spread, when a generalized seizure, with or without a "psychomotor" element, might be the outcome.

Many instances of "psychomotor" clinical attacks arising from unilateral localized cortical areas and from a focus deep in the brain have been given in the body of the paper. All these "psychomotor" clinical episodes differed from one another. Perhaps the one thing in common to all the episodes included under this heading was the occurrence of some disturbance of consciousness, transitory or otherwise.

There are other examples of the wide variation of manifestations that may accompany these slow rhythmic waves. Jasper and associates,[30] for instance,reported bilateral rhythmic slow potentials in the records of a group of behavior problem children, and Putnam and Merritt [31] observed similar wave patterns for patients showing only periods of mental dulness. It appears that the location and the intensity of the disturbance must have played at least a considerable part in determining the variability in the symptoms presented by these different subjects.

Although it seems that rhythmic 3 to 6 per second waves may from time to time appear in almost any high voltage epileptic discharge, it remains to be explained why only a few people have these rhythmic potentials as a prominent feature of their electroencephalograms. It was the few persons with such an electrical pattern in this study who had most pronounced "psychomotor" symptoms, an association pointed out by Gibbs, Gibbs and Lennox.[3b] This was particularly true when the electrical abnormality involved the left temporal lobe or arose deep in the brain.

The So-Called Petit Mal Type of Disturbance.—The 3 per second wave and spike pattern is apparently the electrical abnormality usually, if not always, present during petit mal seizures, as originally reported by Gibbs, Davis and Lennox [2] and corroborated by Jasper and Kershman [4] and

30. Jasper, H. H.; Solomon, P., and Bradley, C.: Electroencephalographic Analyses of Behavior Problem Children, Am. J. Psychiat. **95**:631-658, 1938.

31. Putnam, T. J., and Merritt, H. H.: Dulness as Epileptic Equivalent, Arch. Neurol. & Psychiat. **45**: 797-813 (May) 1941.

others. The few patients whom my associates and I studied during clinical attacks showed typical 3 per second waves and spikes.

Patients with such electrical patterns (subclinical or clinical) apparently have essentially the same type of epilepsy (with respect to the location of the discharge) regardless of whether or not they have a history of clinical petit mal attacks, for in all patients presenting such bilaterally synchronous potentials the origin of the disturbance appears to be in a deep central midline area. When the discharge in such a patient is of a certain intensity, he may become irritable and present the picture of a "behavior problem." When the voltage of the waves shows a decided increase for a brief period, a petit mal seizure may result. On the other hand, if the electrical activity becomes more intense still and is prolonged, a generalized grand mal episode without localizing features results (as pointed out by Jasper and Kershman [4]). Certain persons with subclinical 3 per second waves and spikes have only grand mal attacks, presumably because when the electrical activity increases in intensity sufficiently to produce clinical symptoms, it always does so in an intense and persistent manner. Theoretically, however, these persons could have a petit mal attack if at any time a brief intense discharge should occur.

All these clinical seizures are characterized by their generalized nature, and there is nothing in their clinical pattern to suggest that they may have originated in a localized area of the cerebral cortex. This is in keeping with the hypothesis of the origin of the activity in a deep central midline area (as indicated by the work of Jasper and Penfield and associates [11]).

Such a hypothesis suggests that although a 3 per second wave and spike pattern (in an epileptic patient) does not always mean that he has petit mal epilepsy, it indicates that he has a specific form of epilepsy with respect to the location of the abnormality, and hence the observation of Gibbs, Davis and Lennox [2] is fundamental if one agrees to disagree over terminology.

So-Called Grand Mal Activity.—In the electroencephalograms studied in this investigation it was not possible to determine whether the presence of high voltage fast waves between clinical attacks indicated that the patient had grand mal epilepsy. High voltage potentials at 15 to 50 per second occurred as a subclinical phenomenon in over 5 per cent of the records, but little significance could be attached to this observation since 86 per cent of all the patients included in the series gave a history of grand mal seizures. That subclinical high voltage fast waves are not specific for any form of epileptic attack is, however, indicated by the work of Finley,[17] who reported their presence to be more frequent in association with neurosyphilis and affective psychoses than with grand mal.

This, of course, does not invalidate the observation of Gibbs and his associates that actual clinical grand mal seizures are accompanied by fast potentials of high voltage. This was true for the 3 patients from whom we obtained tracings during a major convulsion.

SUMMARY

The electroencephalograms of 100 patients with recurrent epileptic seizures could be classified with considerable accuracy according to the classification of Jasper and Kershman,[4] which is based on the localization of the discharge. The results of the study are in keeping with the hypothesis that most epileptic discharges (over 65 per cent) apparently arise as local or focal disturbances in the brain. There appears to be a distinct correlation between the location of the electrical abnormality and the type of clinical seizure suffered by the patient, as described by Jasper and Kershman.[4]

It appears that high voltage abnormal waves in the electroencephalograms of epileptic patients commonly (in over 50 per cent of the 65 patients from whom such waves were recorded) take on a rhythmic 3 to 6 per second pattern for brief, or more prolonged, periods. This seems to hold true regardless of whether the waves originate in a focal cortical discharge or in a deeper disturbance. When these 3 to 6 per second slow potentials are of medium voltage and occur relatively infrequently, or are confined to "silent" areas of the brain, there may be no associated symptoms. If they are of high voltage and involve large areas or regions, such as the left temporal lobe, there may result a variety of manifestations, such as "psychomotor" attacks, "psychic equivalent or variant" states or of episodes of "amnesia," as described by Gibbs, Gibbs and Lennox.[3b] At times an apparently similar type of electrical abnormality is seen in the records of behavior problem children, as reported by Jasper and associates [30] or is associated with periods of mental dulness, as observed by Putnam and Merritt.[31] It seems that the electrical pattern or abnormality in all epileptic patients with this slow rhythmic type of disturbance is essentially the same, but is nonspecific, and that the variability in the associated symptoms depends largely on the location and intensity of the discharge.

This hypothesis seems to explain why so-called psychomotor electrical patterns (3 to 6 per second rhythmic slow waves) may be seen in patients who have no history of "psychomotor" clinical manifestations and yet may be associated with such attacks in other persons.

In the present study all patients with a history of episodes classified as of "psychomotor," "psychic equivalent or variant" or "amnesia" type also had focal, petit or grand mal seizures.

Evidence indicates that in a high voltage epileptic discharge high frequencies do not necessarily produce "overactivity" or slow frequencies "underactivity." This observation, as recorded here, applies only to the abnormal waves associated with epilepsy.

In the formation of the slow waves (random or rhythmic in the electroencephalograms of epileptic subjects) it is suggested that, although the periods of activity of the neurons are more or less synchronous (are simultaneous—show hypersynchrony), the resulting composite impulse discharges which presumably contribute to the slow potential changes are asynchronous and often of high frequency. In the case of fast waves (at least when rhythmic), on the other hand, the periods of activity of the neurons, as well as the impulse discharges, may be synchronous.

Dr. Thomas K. Davis, attending psychiatrist, Lenox Hill Hospital, helped to make this work possible, and Dr. Herbert Jasper interpreted some of the records.

THE ELECTROENCEPHALOGRAM ASSOCIATED WITH CHRONIC ALCOHOLISM, ALCOHOLIC PSYCHOSIS AND ALCOHOLIC CONVULSIONS

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Since electroencephalography gives reliable data concerning one type of cerebral activity, this technic has been employed as an aid in the study of alcoholism and the alcoholic psychoses. Loomis and associates,[1] in 1936, studied a patient during an alcoholic stupor and noted increase in the brain potentials and slowing of the rhythm. Gibbs and associates [2] reported that administration of alcohol first increases the voltage of fast frequencies but that later, as consciousness is lost, slow rhythms appear. Rosenbaum and associates [3] noted pathologic brain waves of the type seen in attacks of epilepsy in 4 patients with chronic alcoholism and convulsions.

An important electroencephalographic study of alcoholism was carried out by Davis and her co-workers.[4] In acute intoxication only slight changes occurred in the electroencephalogram, in spite of pronounced changes in mood, deportment and mental acuity; and clearly evident effects, namely, reduction in fast activity and a simultaneous increase in slow activity, occurred only when the level of awareness of the patient was greatly reduced. These authors also studied 15 nonpsychotic subjects who had been confined to a hospital for chronic alcoholism and reported that the "alcoholics appeared to be between the normals and the psychotics in respect to the normality and stability of their electroencephalographic records."

Clinical observations on patients with chronic alcoholism and alcoholic psychoses have led to certain interesting questions. For example, does prolonged excessive indulgence in alcohol lead to abnormalities in the electrical activity of the cortex? Are any electroencephalographic changes associated with the alcoholic psychoses? Does alcoholic deterioration or Korsakoff's psychosis result in a peculiar electroencephalographic pattern? Is there an epileptoid component in pathologic intoxication, and is the electroencephalogram an aid in detecting this? Finally, in patients with epilepsy associated with alcoholism ("rum fits") does the electroencephalogram show the dysrhythmias which have been described as characteristic of idiopathic epilepsy?

We have studied the electroencephalograms and clinical features of a series of patients with chronic alcoholism with and without psychosis and have paid special attention to epilepsy in association with chronic alcoholism; on the basis of this study we are able to give preliminary answers to some of these questions.

RECORDING AND CLASSIFICATION OF ELECTROENCEPHALOGRAMS

Electroencephalograms were recorded with a Grass six channel amplifying system. Electrodes were applied to both frontal, both parietal and both occipital areas. Both monopolar and bipolar recordings were taken; for the former, interconnected leads from the mastoid served as an indifferent pole. Bipolar leads proved helpful in eliminating muscle artefacts in tense and restless patients. The tracings were obtained with the patient in a recumbent position, with the eyes closed, in a darkened and quiet room.

The records are classified as normal, borderline or abnormal on the basis of experience with hundreds of normal and abnormal subjects. A normal record is defined as one in which the brain wave rhythm has a frequency of between 8 and 12 per second and in which the pattern is unchanged, or only slightly modified, by two minutes of hyperventilation. An abnormal record is defined as one in which the prevailing activity is outside the frequency range of 8 to 12 per second, or one which shows a decided change in the character of the pattern as a result of two minutes of overbreathing. Borderline records are those without a dominant frequency pattern or with characteristics of both normal and abnormal tracings. Certain subjective factors are of necessity involved in the classification of electroencephalograms, but in essence our classification of normal and abnormal records agrees with that of other investigators in the field.

From the Department of Psychiatry, Harvard Medical School, and the Boston Psychopathic Hospital, Dr. Harry C. Solomon, Director.

1. Loomis, A.; Harvey, E. N., and Hobart, G.: Electrical Potentials of the Human Brain, J. Exper. Psychol. **19**:239-249, 1936.

2. Gibbs, F. A.; Gibbs, E. L., and Lennox, W. G.: Effects on the Electroencephalogram of Certain Drugs Which Influence Nervous Activity, Arch. Int. Med. **60**: 154-166 (July) 1937.

3. Rosenbaum, M.; Murray, L.; Philip, P., and Goldman, D.: Convulsive Seizures in Delirium Tremens, Arch. Neurol. & Psychiat. **45**:486-493 (March) 1941.

4. Davis, P. A.; Gibbs, F. A.; Davis, H.; Jetter, W. W., and Trowbridge, L. S.: The Effects of Alcohol upon the Electroencephalogram, Quart. J. Stud. on Alcohol **1**:626-637 (March) 1941.

MATERIAL

A series of 157 patients with chronic alcoholism admitted to the Boston Psychopathic Hospital over a five year period, from 1939 to 1944, formed the basis of the investigation. In most instances only one electroencephalogram was taken for each patient, but for some of the patients repeated electroencephalograms were obtained, either during a single period of hospitalization or during successive admissions. The diagnostic classification of the type of alcoholic disorder from which the patient was suffering was decided after careful scrutiny of the hospital record. Special attention was given to the patients with epilepsy associated with alcoholism and to the clinical status of these patients on the day that the electroencephalogram was taken.

The electroencephalographic controls for these records of patients with chronic alcoholism consisted of tracings obtained from a series of 240 nurses, physicians, medical students and hospital personnel, between the ages of 18 and 40; 90 per cent of the control subjects were between 20 and 30 years of age. The percentage of abnormal electroencephalograms in this group was 10 per cent.

As an added control for the electroencephalograms of our patients with epilepsy associated with alcoholism ("rum fits") who had onset of seizures relatively late in life (at an average age of 35), the tracings were compared with the electroencephalograms of 115 patients with idiopathic epilepsy with an average age of onset of convulsions at 35. The records of the latter group were made available to us through Dr. and Mrs. F. A. Gibbs, from their collection of over 1,000 records of epileptic patients.

DATA AND RESULTS

In an analysis of the factors which may conceivably affect the incidence of abnormalities in the electroencephalogram in any heterogeneous group of patients, the age of the patients and the type of clinical disorder from which they suffer are of great significance.

In a recent study by one of us,[5] the effect of age and the clinical condition on the electroencephalographic abnormalities of a large group of neuropsychiatric patients was so striking that it has become imperative to analyze the electroencephalograms for all types of patients with these two factors in mind. We found that 24 per cent of our total group of 157 patients with alcoholic disorders had abnormal electroencephalograms. However, when the incidence of abnormalities was considered with relation to age (table 1), only 5 per cent of abnormal records was found in the age group from 20 to 30, whereas an incidence of 18 per cent was noted for the age group from 30 to 40, 19 per cent for the age group from 40 to 50 and 48 per cent for the age group of 50 and above. This trend of an increasing incidence of abnormalities in the older age groups is similar to that shown for the neuropsychiatric disorders in general and for several of the clinical diagnostic subgroups, for example, involutional melancholia, psychoneurosis and psychopathic personality.[2] In our control group, made up of nurses, physicians, medical students and hospital personnel, the majority were between 20 and 30 years of age, and the incidence of abnormalities was 10 per cent. For the same age range, therefore, the alcoholic patients had an incidence of abnormal electroencephalograms of 5 per cent and the normal subjects an incidence of 10 per cent, a difference which is not significant (table 1).

TABLE 1.—*Relation of Age to the Incidence of Electroencephalographic Abnormalities*

Age Group, Yr.	Number	Percentage with Electroencephalographic Abnormalities
Patients with Chronic Alcoholism		
20 to 30	21	5
30 to 40	58	18
40 to 50	52	19
50+	26	48
Control Subjects		
20 to 30	240	10

The type of disorder from which the patient suffers is of major importance. Since, for the most part, there are no fundamental physiologic or pathologic differences on which to base a classification of the alcoholic disorders, one is forced to resort to classification by clinical syndromes, which involves necessarily many subjective elements of interpretation. After careful consideration of the histories of the patients and the elimination of certain doubtful or unsatisfactory cases, we agreed on a workable scheme,

TABLE 2.—*Relation of Clinical Classification of Alcoholic Psychoses to the Incidence of Abnormal Electroencephalograms*

Clinical Diagnosis	Number of Patients	Abnormal Electroencephalograms	
		Number	Percentage
Chronic alcoholism without psychosis	55	3	5
"Rum fits" (average age of onset, 35)	24	4	17
Alcoholic hallucinosis or delirium tremens	34	7	21
During active disease	(13)	(6)	(46)
After recovery (patient asymptomatic)	(21)	(1)	(5)
Undiagnosed psychosis with chronic alcoholism	24	8	35
Pathologic intoxication	5	3	..
Korsakoff's psychosis	4	3	..
Alcoholic deterioration	11	9	..
Total	157	37	24
Controls	240	24	10
Idiopathic epilepsy (average age of onset of seizures, 35)	115	86	75

5. Greenblatt, M.: Age and Electroencephalographic Abnormality in Neuropsychiatric Patients, Am. J. Psychiat. **101**:82-90 (July) 1944.

which nevertheless required in many instances an arbitrary diagnosis. Table 2 shows the diagnostic classification used, the numbers of patients in each category and the percentage of abnormal electroencephalograms obtained in each class. Also shown are the two control groups: 1. A group of 240 persons normally adjusted in the community who may serve as a base line, or norm, for all other diagnostic categories or subdivisions. Of this number, 10 per cent had abnormal electroencephalograms. 2. A group of 115 patients with essential or idiopathic epilepsy whose seizures began relatively late in life, at an average age of 35. This group should be compared with the group of patients with "rum fits," whose seizures also appeared relatively late in life, at an average age of 35. In table 2 the alcoholic disorders are arranged in order of rank, that is, in terms of the ascending incidence of abnormal electroencephalograms.

Chronic Alcoholism Without Psychosis.—It is interesting to note that in this group only 5 per cent of the electroencephalograms were abnormal. The tracings were taken when the period of acute intoxication responsible for the admission to the hospital was over and when the patient was clinically free of symptoms. Chronic alcoholism without psychosis, essentially the least complicated of the alcoholic disorders from the standpoint of formation of symptoms, is associated with the lowest percentage of electroencephalographic abnormality of any of the alcoholic syndromes. In fact, the percentage of abnormal electroencephalograms in this series was less than that encountered among control subjects normally adjusted in the community. Much has been written concerning the personality "defects" which are supposedly present in persons who readily establish the habit of dependence on alcohol. We have no electroencephalographic evidence that these patients exhibit abnormal cortical function. There is also no evidence that the duration of chronic alcoholism has any significant relation to the amount of electroencephalographic abnormality. Normal records were obtained from persons with a long history, as well as from those with a relatively short period, of heavy indulgence.

"Rum Fits."—Of the 24 patients with "rum fits," only 4 (17 per cent) had abnormal electroencephalograms, a surprising finding in view of the high percentage of abnormal records obtained for almost all other patients who had convulsions as an outstanding symptom. These patients will be discussed in more detail later.

Alcoholic Hallucinosis or Delirium Tremens.—A total of 34 patients had a condition diagnosed as alcoholic hallucinosis or delirium tremens, and a total of 7, or 21 per cent, had abnormal electroencephalograms. However, there was a definite correlaton between the presence of active symptoms and electroencephalographic abnormality. Thirteen patients in the group were studied during active hallucinosis or delirium, and 6 of these, or 46 per cent, had abnormal electroencephalograms. On the other hand, 21 patients were studied after recovery from hallucinosis or delirium tremens, and only 1, or 5 per cent, had an abnormal record.

The data suggest that an abnormal electroencephalogram is related to active disease and that clinical recovery is associated with improvement in the pattern. An abnormal record is evidently not a prerequisite to the appearance of hallucinosis or delirium in a patient with chronic alcoholism. Although it seems likely, we have as yet no data to indicate that an abnormal electroencephalogram indicates greater susceptibility to hallucinosis or delirium tremens after heavy indulgence. However, an abnormal tracing obtained during the acute stage of disease is often indicative of a longer clinical course than is a normal one.

Undiagnosed Psychosis with Chronic Alcoholism.—In this group were placed 24 chronically alcoholic patients with psychosis of a relatively undifferentiated type not readily classified in other categories. Patients with many heterogeneous types of conditions were included here, but all were psychotic at the time of the electroencephalographic examination. Thirty-five per cent of the records were abnormal.

Pathologic Intoxication.—Patients in whom small amounts of alcohol produced disproportionately great outbursts of violent, aggressive overactivity with clouding of consciousness were regarded as exhibiting pathologic intoxication. Five patients in this category were studied after recovery from intoxication. Three had abnormal electroencephalograms. Both slow and rapid potentials were present in the abnormal tracings, but no tracing presented the paroxysmal dysrhythmia characteristic of the epileptic disorders.

Korsakoff's Psychosis.—Four patients whose illness was diagnosed as Korsakoff's psychosis were studied electroencephalographically during the period of notable impairment of mental functions; 1 of these was followed until recovery occurred under intensive treatment. Three of the 4 patients, including the patient whose condition later cleared up, had abnormal electroencephalograms. The fourth had a normal record despite a well marked Korsakoff psychosis. The patient who was followed during treatment showed improvement in the electro-

encephalographic pattern associated with clinical recovery.

Alcoholic Deterioration.—Eleven patients presenting the clinical picture of alcoholic deterioration were studied; 9 had abnormal electroencephalograms. However, the average age of these patients (49 years) was higher than that of the patients in the other diagnostic groups, and conceivably the electroencephalographic abnormality could in part be due to the factor of age.

Electroencephalographic Pattern and Prognosis of the Alcoholic Disorders.—The vast majority (80 per cent) of the patients with alcoholism were discharged from the hospital as improved within ten days. The remainder (20 per cent) were transferred to other mental disease institutions for more prolonged care and treatment because of the greater severity and chronicity of their illness. Fifty per cent of the patients who had abnormal electroencephalograms and only 11 per cent of the patients with normal electroencephalograms required more prolonged hospitalization. The abnormal electroencephalographic pattern was correlated not only with the presence of active hallucinosis, delirium and confusion but with a longer clinical course.

ALCOHOLISM AND EPILEPSY

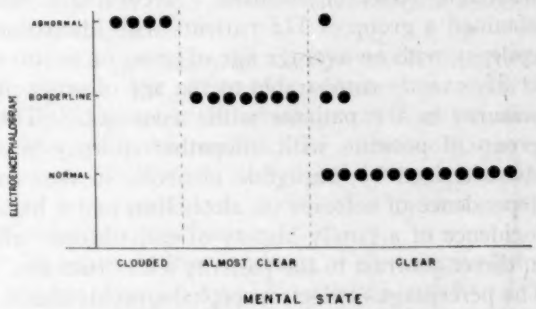
When convulsions and chronic alcoholism appear together in the same patient, it is often a difficult problem to determine their precise etiologic relation. A history taken from the patient is often grossly unreliable because these patients have a tendency to obscure the relation between the two factors; for example, they date the convulsions before the onset of alcoholism or emphasize that seizures do not occur during the height of alcoholic indulgence but have their onset after the indulgence has ceased. When the latter claim is made, one must be suspicious of a primary withdrawal phenomenon, for sometimes there is a short lag between the cessation of drinking (often spontaneous, because of gastritis and vomiting) and the appearance of seizures.

A logical classification on the basis of the relation between convulsions and alcoholism would have to do justice to the many possible gradients of etiologic dependence of seizures on alcoholic indulgence. At one extreme, there would be patients who never had convulsions save at the height of alcoholism or shortly after abrupt withdrawal. Such a condition is already clinically well recognized and is given the name "rum fits," or "whisky fits." At the other extreme, there would be patients suffering from essential or idiopathic epilepsy with or without alcoholic indulgence, but with no manifest etiologic relation between the two. Between these two extremes there would be patients with a tendency to epilepsy in whom some indulgence in alcohol would at times be required to bring out the underlying predisposition.

We approach the problem by studying electroencephalographically and clinically the two extremes: (*a*) patients with "rum fits," with a clearcut etiologic relation between alcoholism and convulsions, and (*b*) patients with idiopathic epilepsy with insignificant indulgence in alcohol and with seizures temporally unrelated to alcoholic intake.

"Rum Fits."—Twenty-four patients, all males, were classified as having "rum fits." Their ages ranged from 26 to 52, with an average of 39. The duration of seizures ranged from days to years, with an average of four years. The duration of heavy alcoholism ranged from one-half year to twenty-seven years, with an average of fourteen years.

An average of ten years of heavy drinking occurred before the first admission to the hospital. Twenty-two of the patients had seizures



Relation between electroencephalographic classification and mental status of patients with "rum fits." Twenty-six electroencephalographic records were taken on 24 patients. The large circles represent one electroencephalogram each.

typical of grand mal attacks. The attacks of 1 patient were described as "fainting spells," and those of another consisted of clonic movements without loss of consciousness.

The histories of these patients were peculiar in that none had convulsions in youth or prior to the onset of heavy drinking. The earliest age of onset of seizures was 26 and the average age of onset 35. Not one of the patients had a family history of epileptic disorder.

The majority of patients with "rum fits" sought admission to the hospital because of postconvulsive clouding of consciousness, often accompanied by excitement. A few patients were followed during recovery from the clouded state, and it was evident that electroencephalographic abnormality disappeared as the clouded

mental state improved. When the type of electroencephalogram for each patient was compared with the mental status of that patient, it appeared that the more clouded the mental picture at the time of the recording the more abnormal was the electroencephalogram (fig. 1).

Of 24 patients with "rum fits," only 4, or 17 per cent, had abnormal electroencephalograms; 3 of these 4 patients had clouding of consciousness, and the fourth was actively hallucinated at the time of the recording.

Idiopathic Epilepsy.—Because of the surprising observation that "rum fits" were associated with little electroencephalographic abnormality whereas almost all other conditions in which convulsions are an outstanding feature are associated with a high incidence of electroencephalographic abnormality, we decided to compare our observations on patients with "rum fits" with those on patients with idiopathic epilepsy. We were especially interested in controlling the age of onset of seizures because of the possibility that a low incidence of electroencephalographic abnormalities might appear in any group of patients with late onset of seizures. Accordingly, we obtained a group of 115 patients with idiopathic epilepsy with an average age of onset of seizures at 35, exactly comparable to the age of onset of seizures in the patients with "rum fits." The group of patients with idiopathic epilepsy was characterized by negligible alcoholic intake, no dependence of seizures on alcoholism and a high incidence of a family history of convulsions—all in direct contrast to the patients with "rum fits." The percentage of electroencephalographic abnormalities for the patients with idiopathic epilepsy was 75 per cent. A consideration of the relation of electroencephalographic abnormality to age of onset of epilepsy in patients with the idiopathic type showed that from youth to old age there was a decrease of 10 to 15 per cent in the incidence of electroencephalographic abnormalities. At comparable ages for onset of seizures, however, the incidence of abnormal electroencephalograms was strikingly higher for idiopathic epilepsy than for "rum fits."

COMMENT

Many investigations have established the fact that a high incidence of electroencephalographic abnormality is associated with the convulsive disorders and that the most significant feature is the paroxysmal dysrhythmia, so thoroughly studied by Gibbs and Lennox. The actual percentage of abnormal electroencephalograms varies with the individual series, but most workers agree that between 60 and 90 per cent of patients with seizures have abnormal electroencephalograms. However, there is always the irreducible minimum who do not show abnormalities despite well substantiated convulsions. To a certain extent, this may be explained on the basis of the severity and frequency of the epileptic attacks —that is, patients with rare or infrequent seizures may show considerably less electroencephalographic abnormality than those with frequent or severe seizures. Age is another factor that may determine the amount of abnormality. There is evidence to indicate that the older the epileptic patients, the lower the percentage of electroencephalographic abnormality.[6]

The question arises whether persons without onset of seizures in childhood and without a family history of epilepsy (that is, without evidence of an inborn epileptic component), yet with seizures due to some other factor, have significant electroencephalographic abnormalities.[7] Only a few observations bearing on this question have been reported. A case of convulsions following abrupt withdrawal of barbiturates with a normal electroencephalogram has been reported.[8] We have observed several such cases of seizures following withdrawal of barbiturates in addicts who were not known to be epileptic and whose electroencephalograms were normal when the seizure was over. A similar example is that of a patient subjected to electric shock therapy. In such a patient electroencephalograms are usually normal before shock, and there is a rapid return to normal after the induced seizure subsides. Except in occasional patients subjected to numerous electrically induced fits in whom one sometimes sees prolonged alteration in the electroencephalogram, the ultimate pattern is normal and similar to the preshock pattern.

Our data lead us to believe that patients with "rum fits" have no inherited predisposition to epilepsy and have normal interseizure records despite years of heavy alcoholic indulgence and of seizures induced by alcohol. We believe that there is often confusion between cases of pure "rum fits" and cases of idiopathic epilepsy in which seizures may at times be precipitated by ingestion of alcohol. In the latter the onset of epilepsy usually precedes the alcoholism by some

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time; in addition, the seizures frequently occur in the absence of any known intake of alcohol. In a small series of such cases we found a high incidence of abnormal electroencephalograms comparable to that for idiopathic epilepsy without alcoholism.

In studying the brain waves of patients with convulsions, we found it important to know the exact mental status at the time of the electroencephalographic examination before evaluating the final normality or abnormality of the tracing because any residual confusion following a seizure may be associated with abnormalities which later clear up. This is particularly true of patients with "rum fits," for these patients are often hospitalized because of postconvulsive clouding, and the clouding of consciousness is frequently prolonged.

Not only postconvulsive confusion but active alcoholic hallucinosis is correlated with abnormal electrocortical activity, and often the hallucinosis is present long after the elimination of alcohol is completed. Surely, excessive alcoholism initiates a neurophysiologic change the persistency of which does not depend on a high level of alcohol in the blood.

An arrangement of clinical categories of chronic alcoholism on the basis of an increasing incidence of electroencephalographic abnormality (as in table 2) turns out to be a rough classification of these disorders according to the severity of the clinical picture, the chronicity of symptoms and, probably, the severity and extent of damage to the brain. The lowest incidence of electroencephalographic abnormality is found in cases of chronic alcoholism without psychosis, essentially the least complicated of all the alcoholic disorders from the standpoint of the formation of morbid symptoms. On the other hand, the highest incidence of electroencephalographic abnormality occurs in cases of alcoholic deterioration, an essentially fixed psychosis with features of the organic reaction type. In our small series of patients with Korsakoff's psychosis (an outstanding paradigm of the "organic syndrome") there was a high incidence of electroencephalographic abnormality, and we anticipate eventual corroboration of this observation when a larger series of such patients has been collected. Relatively little abnormality is found for patients with delirium tremens or acute alcoholic hallucinosis, both disorders with rapid recovery. However, with chronic alcoholic hallucinosis the electroencephalogram tends to be more abnormal than with the acute type.

SUMMARY

In a study of the clinical histories and electroencephalograms of 157 patients with chronic alcoholism with and without psychosis, including patients with convulsions due to chronic alcoholism, the following observations were made:

The incidence of electroencephalographic abnormality in patients with chronic alcoholic disorders increases with age.

Persons with chronic alcoholism without psychosis, irrespective of the duration of drinking, show essentially nothing of significance in the electroencephalogram.

Chronic alcoholism with psychosis is in general associated with an incidence of electroencephalographic abnormality which is higher than normal. In patients with alcoholic psychosis the presence of confusion or hallucinations is frequently associated with electroencephalographic abnormality, and the disappearance of hallucinations or confusion is often accompanied by a change toward a more normal electroencephalogram.

No evidence of paroxysmal dysrhythmia was found in 5 patients with pathologic intoxication, although 3 of the 5 patients had abnormal electroencephalograms.

Of the patients with chronic alcoholism with psychosis, the highest incidence of electroencephalographic abnormality was found among those with deterioration or Korsakoff's syndrome. No specific electroencephalographic pattern was found in a group of 4 patients with Korsakoff's psychosis.

A relatively low incidence of electroencephalographic abnormality (17 per cent) was found in a series of 24 patients with "rum fits" (with a negative family history and a negative past history for epilepsy and with seizures occurring only in association with alcoholism). On the other hand, a relatively high incidence of electroencephalographic abnormality (75 per cent) was found in a large series of patients with idiopathic epilepsy with onset of seizures in the same age range as that of the patients with "rum fits."

In spite of the occurrence of seizures, patients with "rum fits" do not have the inborn epileptic predisposition observed in persons with idiopathic epilepsy.

An abnormal electroencephalogram may be of aid in predicting the duration of illness in patients hospitalized for chronic alcoholism.

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THE HYPOTHALAMUS AND AFFECTIVE BEHAVIOR IN CATS

A STUDY OF THE EFFECTS OF EXPERIMENTAL LESIONS, WITH ANATOMIC CORRELATIONS

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Evidence has accumulated which indicates that the hypothalamus may be concerned in certain elements of behavior the total pattern of which appears to be related to the outward manifestation of emotion. Stimulation of the hypothalamus in the region occupied by the medial bundle of the forebrain in the lateral part of the hypothalamus has been observed by Kabat and associates to cause mydriasis, increased respiration, struggling movements with clawing and biting, salivation, horripilation and sweating on the pads of the feet. In addition, cessation of peristalsis was noted, and sometimes evacuation of the bladder (Kabat, Anson, Magoun and Ranson [1]; Kabat, Magoun and Ranson [2]; Ranson, Kabat and Magoun [3]; Kabat,[4] and Kabat, Magoun and Ranson [5]). The points of stimulation were shown to be restricted largely to the lateral hypothalamic area. Similar results were obtained from anesthetized and unanesthetized cats. These reactions were not elicited when the infundibular stalk, the septum pellucidum, the anterior commissure, the thalamus and the internal capsule were stimulated with a current of the same intensity. The same general pattern of responses suggestive of rage had been observed by other investigators, but the precise area of stimulation had not previously been accurately localized (Karplus and Kreidl [6]). Dusser de Barenne and Sager [7] observed that when a small amount of strychnine was injected into the hypothalamus, cats displayed intense activity resembling sham rage; they hissed when approached and showed horripilation, mydriasis, salivation, defecation and urination. Strychninization of the thalamus caused hypersensitivity of the skin to temperature, touch and pain stimuli, but the cats did not show the affective responses observed on strychninization of the hypothalamus.

Somewhat similar results for electrical stimulation have been reported with human subjects. Grinker and Serota [8] stated that transsphenoidal stimulation by means of an electrode pushed into the nasal surface of the sphenoid bone about the middle of the floor of the sella turcica produced the usual general visceral effects: acceleration of the respiratory and the heart rate, dilatation of the pupils, elevation of blood pressure, increase in vasomotor tone, rise in body temperature, sweating and contraction of the bladder. Additional effects were reflected in the subjective and overt emotional reactions of the patients, e. g., profound anxiety and uncontrollable sobbing (Grinker and Serota [8]; Grinker [9]). All of the patients responded with

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expressions of fear, and, although none showed anger, stronger stimuli elicited tonic, and sometimes clonic, movements of the extremities comparable to the running and clawing movements reported for cats.

Mechanical stimulation of the floor of the third ventricle has also been reported as effective in producing outbursts of manic reactions in human subjects (Foerster and Gagel [10]). Singing, expressions of terror and rage, and general hyperactivity have been noted when this region was swabbed or disturbed during removal of pathologic growths with local anesthesia (Clark, Beattie, Riddoch and Dott [11]). However, White [12] found that in man mechanical and electrical stimulation of the hypothalamus produced bradycardia, a rise in blood pressure and a tendency to drowsiness or coma. He noted no alterations in respiration, nor could he detect any psychic or sensory changes.

Observations on emotional responses in man in clinicopathologic studies, while not as precise in the localization of the definite areas or nuclei concerned as is possible with animal experimentation, are nevertheless valuable in consideration of the possible anatomic correlates of emotional behavior. Practically all the disturbances consequent to lesions in and stimulation of the hypothalamus in experimental animals have been reported in man after trauma, operative manipulation, tumors, vascular lesions and infections of the hypothalamus. The behavior of the patients reflected a subjective phase, as well as the outward manifestations of emotion, some of them giving evidence of hallucinations, flight of ideas, terror, rage and anxiety. The behavior of others indicated pleasanter moods, which might be witty, jocular or obscene (Foerster and Gagel [10]; Cox [13]; Alpers [14]; Weisenburg [15]; Clark and associates [11]; Claude and Lhermitte [16]; Fulton and Bailey [17]; Cushing [18]).

Modern experiments attempting to delimit the various parts of the brain concerned with emotional expression through lesions and ablations were given their original impetus by Goltz [19] in 1892. He observed that a decorticate dog with injury to the corpora striata and the thalamus occasionally displayed emotional hyperexcitability suggestive of rage in response to innocuous stimuli. Similar results were obtained by other investigators with decorticate cats and dogs: Dusser de Barenne,[20] with decorticate cats, and Rothmann,[21] with a decorticate dog. Bard [22] and Bard and Rioch [23] made an exhaustive study of the effects of decortication on emotional expression in cats. They expressed the belief that the "sham rage" displayed by their experimental animals was due to the release of subcortical levels from cortical inhibition. The somatic component, or at least parts of the somatic component, of rage may possibly be elicited after all brain substance rostral to the mesencephalon is removed, but the full sham rage reaction requires that a portion of the posterior hypothalamus remain. This work indicates that the area most important for the full expression of

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rage is the caudal part of the hypothalamus. The phenomenon of "sham rage" has also been reported in man (Wortis and Maurer [24]).

Attempts to determine areas of the cortex specifically concerned in such a release phenomenon have not been consistently successful. Spiegel, Miller and Oppenheimer [25] reported that of 8 cats in which the frontal lobes were destroyed rostral to the sigmoid gyrus, only 1 showed definite rage reactions. Furthermore, Magoun and Ranson [26] removed the portions of the hemispheres rostral to the ansate sulcus in 8 cats, and none of the animals displayed the intense emotional hyperexcitability of sham rage, nor did these cats display any of the cataleptic symptoms that Barris [27] had reported to occur in cats after ablation of the same general area. Spiegel, Miller and Oppenheimer [25] observed that the production of lesions encroaching on the olfactory tubercle or in the fornices was followed by rage reactions. The most pronounced rage effects were observed in animals in which the lesions were said to affect the fornix, with involvement of the adjacent septum pellucidum. These authors also reported the appearance of rage reactions, and occasionally of cataleptic symptoms, following bilateral injury to the amygdaloid nuclei. No rage behavior followed elimination of the following regions: the sigmoid and coronal gyri; the lateral surface of the parietal lobe; the medial surface of the gyrus marginalis; the lateral surface of the temporal lobe, and the lateral, as well as the medial, surface of the occipital lobe.

Lesions of the hypothalamus itself in experimental animals have also been observed to be associated with changes in affective behavior. Bilateral lesions in the caudal part of the hypothalamus produce somnolence and catalepsy in cats, together with a decline in emotional reactivity (Ingram, Barris and Ranson [28]). Probably the most striking emotional changes were noted in wild monkeys, which became tame and showed no fear or anger after the placement of lesions dorsolateral to the mamillary nuclei (Ranson [29]). Kessler [30] found that unilateral destruction of the hypothalamus or of the ventromedial portion of the thalamus in cats resulted in positive rage reactions.

Ingram [31] frequently observed notable changes in "personality" in certain cats with bilateral hypothalamic lesions. These animals, formerly tame and friendly, became savage and would attack man with little hesitation. This picture was exactly the reverse of that noted in cataleptic animals. The lesions in these savage cats were usually in the medial portions of the hypothalamus.

The work to be reported in this paper was concerned with the following problems: (1) a study of changes in behavior in cats following production of lesions in various parts of the hypothalamus, with lesions in other regions of the brain as controls; (2) correlation of such changes in behavior as might be associated with the anatomic alterations caused by the destruction of nerve tissue, and, especially, an attempt to determine whether reactions of a savage type may follow destruction of any specific structural entities in the hypothalamus. In addition, various areas of other parts of the brain were destroyed or removed in 11 cats, not only to determine whether such lesions would ameliorate or intensify rage reactions in savage cats but to produce rage reactions in cats which had not become savage.

MATERIAL AND METHODS

In this experiment 42 cats were selected from several hundred on the basis of friendliness and failure to show aggressive behavior even when subjected to nociceptive stimuli. Only 1, a female cat, P36, showed the slightest tendency to become aggressive when roughed; otherwise, she was friendly and affectionate, and even when she showed such aggressiveness, she would respond immediately to friendly overtures with purring and other signs of feline friendliness and affection. The behavior of these animals was studied daily for several weeks before the placing of the hypothalamic lesions.

The cats were operated on while under anesthesia induced with pentobarbital sodium, and lesions were placed in the hypothalamus by use of the Horsley-Clarke stereotaxic instrument. Careful asepsis was maintained. Postoperative observations were made daily over periods ranging from several weeks to more than a year. Eleven of these cats were subsequently

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subjected to the placing of various cortical lesions and ablations, and day by day records of their postoperative behavior were made.

An attempt was made by special means to arrive at a reasonably objective appraisal of the behavior of the animals when subjected to nociceptive stimuli. The animal to be studied was harnessed in such a way that two electrodes connected with a Harvard inductorium were in contact with the skin of the dorsal region. This harness did not interfere with freedom of movement. The cat was placed in a large cage arranged so that the observer was out of the animal's range of vision. The cage was equipped with noise makers and air blasts under remote control. Each cat was placed in the cage once prior to the operation and its behavior studied and recorded in terms of response to raucous noises, air blasts and shocks of faradic current of controlled intensity. After recovery from the operation, the animal was harnessed and placed in the cage repeatedly, but without being subjected to nociceptive stimuli. This was done to eliminate or minimize any conditioning effect. Later, responses to shocks and noises were noted, in an attempt to determine whether any changes in behavior response could be detected. This special method of study was a supplement to continual daily observation of the general attitudes and behavior of the cats in regard to contact with other animals and with human beings and in response to handling, feeding, and the like.

Motion picture records of the general behavior of the cats before and after operation were also made.

When the behavioral studies were concluded, the cats were killed, their heads perfused with a 10 per cent concentration of neutral solution of formaldehyde U. S. P. and the brains removed. After further fixation in the solution of formaldehyde, the diencephalic regions were dehydrated and embedded in pyroxylin.

Sections 50 microns thick were cut; every other section was stained with cresyl violet for cells, and the alternate sections, by the Weil method for myelin sheaths. In addition to the specimens from the 42 cats in the experimental series, there were available 28 brains from cats in which hypothalamic lesions had been placed for other purposes. Twenty-three of the animals in the latter group had become irritable or savage after operation. Five, however, had especially good behavior, despite the hypothalamic lesions, and were an important addition to the controls already available. Sections of their brains were also prepared. The 70 cat brains were studied microscopically, and attempts made to correlate behavioral changes with the location and extent of the injury to the brain.

Of the whole series, 16 specimens were from cats showing no postoperative change in behavior, in spite of the presence of hypothalamic lesions. These, and a group of animals subjected to "dummy" operations, constituted controls.

The operations for removal of cortical tissue were carried out by direct exposure and careful visualization of the areas to be excised. Careful asepsis was maintained; all bleeding was controlled, and the operations were performed with minimum trauma to areas not removed. During the postoperative period, fluids or food was forced as indicated by the condition of the animal.

GENERAL CHANGES IN BEHAVIOR

In this work only those outward manifestations which man has learned to associate with normal expressions of emotion in cats were studied. The subjective significance of these physical expressions can only be inferred. Bard [22d] has aptly expressed this viewpoint:

> . . . It is doubtless impossible to determine whether a cat standing up to an attacking or threatening dog is subjectively experiencing rage or fear or a mixture of both, but on the supposition that different things should have different names it can be asserted that a normal cat is capable of displaying both rage and fear. When as a result of painful or rough treatment or in response to attack by another animal a cat reacts by spitting and aggressive biting and clawing, it is proper to call it one thing. When in response to similar stimuli or to other happenings, especially a sudden and unexpected loud noise, this same cat dashes off in a furtive manner, mewing plaintively, and tremblingly goes to cover on the first opportunity, it is necessary to designate this as something else. General usage leads one to call the former an expression of anger, the latter a display of fear or terror. It is significant that when observers of varied experience are shown these reactions by means of motion pictures in which the stimulus is not revealed they are unanimous in characterizing the one as a display of rage, the other as an exhibition of fear. This disposes of any possibility that knowledge of the stimulus plays an important part in determining the name given the behavior.

The behavior patterns of the cats in this experiment were not always so clearly cut. Some exhibited the same friendly behavior after operation as before. At the other extreme, some animals, when in the presence of the observer, continually showed all the physical manifestations which have been observed in normal cats when provoked to rage, i. e., mydriasis, horripilation, defecation, urination, vocalization of anger, spitting, lashing of the tail and a kyphotic attitude. They would attack the observer in a vicious, aggressive manner, biting and clawing, when attempts were made to pet them or when other innocuous stimuli were applied.

The exact degree of change in the intermediate types of behavior was often difficult to ascertain. In this account these postoperative reactions have been divided arbitrarily into four classes and are indicated as such in tables 2 to 4. These categories may be summarized as follows: (1) slight change, represented by animals usually harmless and friendly to a moderate degree, but with a tendency to use teeth or claws in escaping painful stimuli; (2) variable behavior, represented by cats which responded favorably to petting but often became savage when roughed, at such times showing many of the physical manifestations of rage; (3) definite and pronounced change, shown by cats which would not respond favorably to petting and displayed signs of rage when approached by the observer, but failed to present one or more of the physical manifesta-

tions, such as urination or defecation, displayed by a savage cat under similar circumstances, and (4) a mixture of poorly directed rage and general psychopathic manifestations, shown by a single animal, D187. This cat showed some motor defect, as indicated by ataxia and stiffness of gait. She had a sullen appearance, frequently panted and made useless movements.

The manner of development of the savage type of behavior varied. In general, a lack of favorable response to petting and fondling was one of the first changes noted. In some cats the complete pattern of savage behavior developed almost immediately or in a very short time after operation. In other cats several days to several weeks elapsed before all the physical manifestations of savageness were displayed. The amount and type of handling undoubtedly were important factors affecting this change, and every attempt was made to subject each of the 42 cats to the same treatment. Often, savage cats can be touched, or even pushed aside, just before they are fed and they are not so likely to attack at such times, especially if they see food. No other inducement produced this effect.

Records of weight were kept on these cats, more as a means of checking on their general health than of observing changes referable to obesity. Several cats which became savage showed proportionately greater increases in weight after the production of the lesions than did the animals which remained normal or those which showed varying degrees of change in behavior. Although the fat, protein and carbohydrate content of the food was not determined quantitatively, all the cats were fed equal portions of the same food. The principal difference in food intake was the rapidity of ingestion. The savage cats either ate hurriedly or wolfed their food, while normal cats given the same diet ate slowly, frequently taking several hours to consume their allowance. No attempt was made to keep measured records of the amount of activity before and after the placement of the lesion; therefore, comprehensive conclusions as to the mechanism producing this obesity are impossible (Hetherington and Ranson [32]; Tepperman, Brobeck and Long [33]). There exists the interesting possibility that the avid appetite observed in certain of the cats was part of a further behavioral change; thus, Brobeck, Tepperman and Long [34] stated that the obesity in rats following hypothalamic lesions was due to "hypothalamic hyperphagia." It is certain that voracious, tigerish appetite and manner of eating were components of the behavior of many of the savage cats in my series, and 1 savage animal kept for many months on an unrestricted diet became exceedingly fat.

BEHAVIOR IN RESPONSE TO CONTROLLED STIMULI

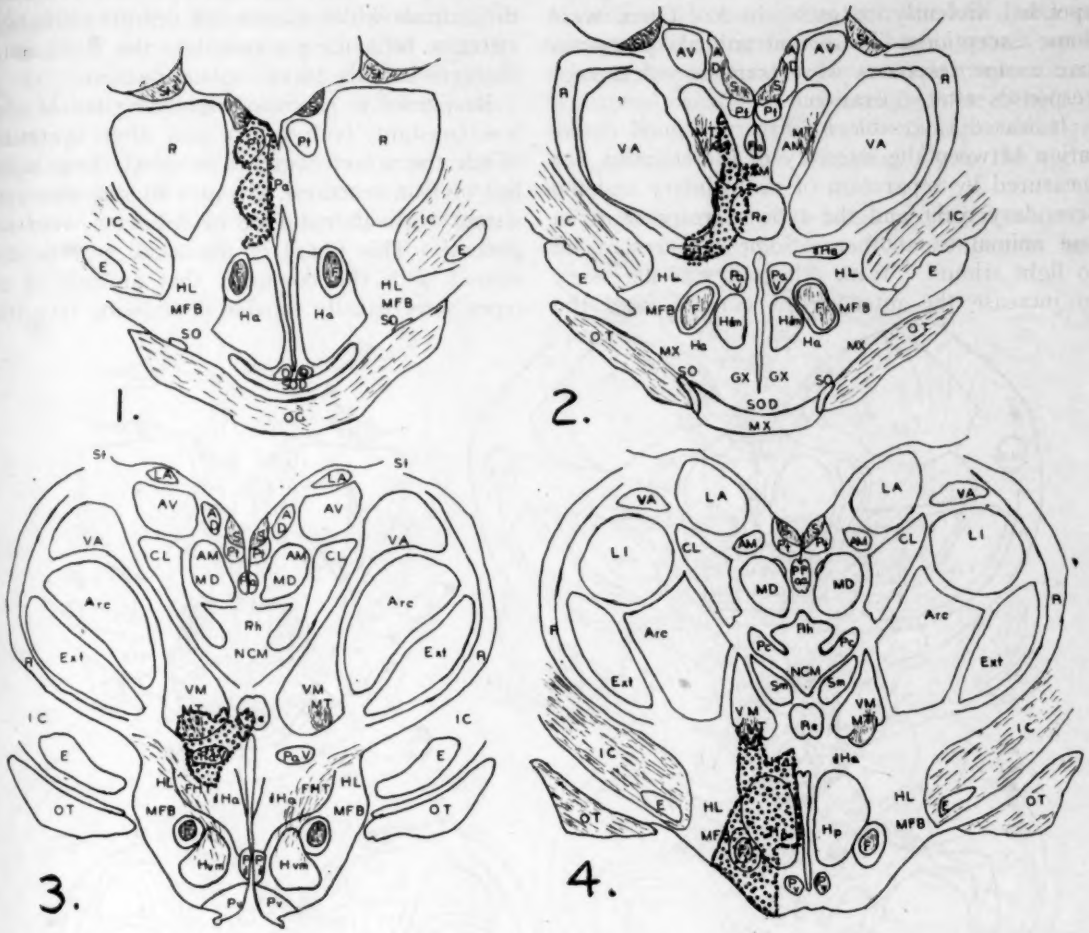
Three general types of behavior were noted in the preoperative responses to electrical shock (recorded in vertical column B in tables 1 to 4): (1) responses in which the cat merely assumed a crouched position and a resigned attitude, with an occasional plaintive meow or light growl; (2) responses in which the cat attempted to escape or fought the harness or attacked some inanimate object in the cage, and (3) responses to the first shock by convulsive leaping and falling until exhaustion occurred. In these observations each cat was given at least fifty shocks with an interrupted faradic current from a Harvard inductorium. The current was of sufficient strength to cause a noticeable contraction of the shoulder muscles. In column B, tables 1 to 4, a response of the first type is indicated by 0. Escape reactions are indicated by *E*, and the number of attempts at escape during the course of the fifty stimulations is shown by the numeral following the corresponding *E*. Aggressive activity is indicated by *R*, and the number of such responses, by the numeral following the corresponding *R*. The convulsive type of reaction is indicated by *C*. When not responding with fear, rage or convulsions, the cat usually sat in a crouched position; sometimes it wandered around meowing or growling, but only definite attempts to escape or vicious attacks were counted during the experiment.

After recovery from the operation and after the treatment to minimize conditioning, as previously described, the cats were again subjected to this type of stimulation under conditions similar to those imposed before operation. The same three types of behavior patterns were observed; these are indicated in column A of the tables in the same manner as the reactions in column B. The principal difference noted between the preoperative and the postoperative reactions was the increased incidence of escape and rage or attack reactions in most of the cats which showed pronounced behavior changes or which became savage. In fact, nearly

32. Hetherington, A. W., and Ranson, S. W.: The Spontaneous Activity and Food Intake of Rats with Hypothalamic Lesions, Am. J. Physiol. **136**:609, 1942.

33. Tepperman, J.; Brobeck, J. R., and Long, C. N. H.: A Study of Experimental Hypothalamic Obesity in the Rat, Am. J. Physiol. **133**:488, 1941.

34. Brobeck, J. R.; Tepperman, J., and Long, C. N. H.: Experimental Hypothalamic Hyperphagia in the Albino Rat, Yale J. Biol. & Med. **15**:831, 1943.



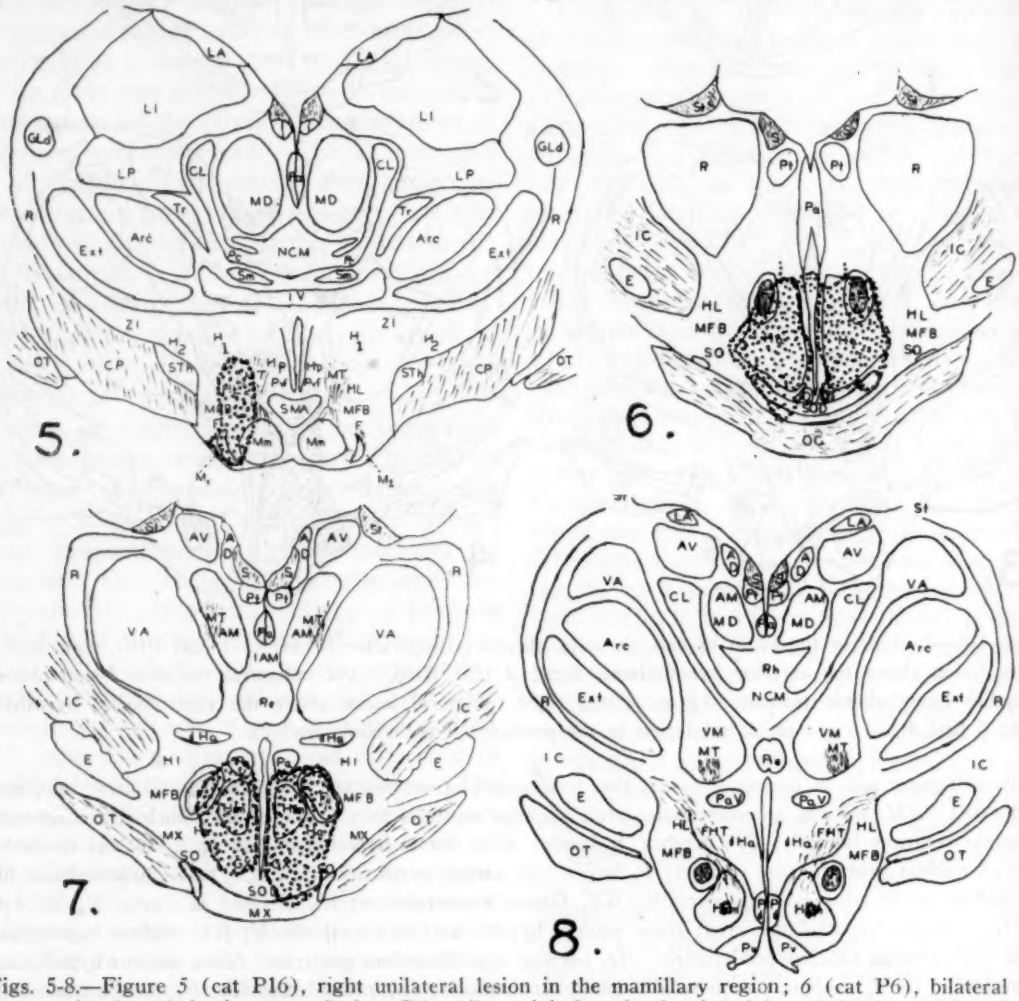
Figs. 1-4.—Lesions in the brain of cats showing normal postoperative behavior. *1* (cat P16), right unilateral thalamic lesion above the anterior hypothalamic area; *2* (cat P16), right unilateral thalamic lesion above the dorsomedial hypothalamic nucleus; *3* (cat P16), right unilateral lesion above the ventromedial hypothalamic nucleus; *4* (cat P16), right unilateral lesion in the posterior hypothalamic nucleus.

In these figures and in figures 9 to 21, the diencephalic structures are designated as follows: *AD*, nucleus anterodorsalis; *AM*, nucleus anteromedialis; *Arc*, nucleus ventralis pars arcuata; *AV*, nucleus anteroventralis; *CL*, nucleus centralis lateralis; *CP*, cerebral peduncle; *dHa*, dorsal hypothalamic area; *E*, nucleus entopeduncularis; *Ext*, nucleus ventralis pars externa; *F*, fornix and nucleus perifornicalis; *FHT*, pallidohypothalamic fibers; *GLD*, nucleus geniculatus lateralis dorsalis; *GX*, Ganser's commissure; H_1, H_1 field of Forel; H_2, H_2 field of Forel; *Ha*, anterior hypothalamic area; *Hdm*, nucleus hypothalamicus dorsomedialis; *HL*, nucleus hypothalamicus lateralis; *HP*, tractus habenulopeduncularis; *Hp*, nucleus hypothalamicus posterior; *Hvm*, nucleus hypothalamicus ventromedialis; *IAM*, nucleus commissuralis interanteromedialis; *IC*, internal capsule; *IV*, nucleus commissuralis interventralis; *LA*, nucleus lateralis pars anterior; *LI*, nucleus lateralis pars intermedia; *MD*, nucleus medialis dorsalis: *MFB*, medial forebrain bundle; *Ml*, nucleus mamillaris lateralis; *Mm*, nucleus mamillaris medialis; *MT*, mammillothalamic tract; *MX*, Meynert's commissure; *NCM*, nucleus centralis medialis; *O*, nucleus suprachiasmaticus; *OC*, optic chiasm; *OT*, optic tract; *Pa*, nucleus paraventricularis anterior; *PaV*, nucleus hypothalamicus paraventricularis; *Pc*, nucleus paracentralis; *Pd*, nucleus hypothalamicus periventricularis dorsalis; *Pt*, nucleus parataenialis; *Pv*, nucleus periventricularis arcuatus; *Pvf*, periventricular fibers; *R*, nucleus reticularis; *Re*, nucleus reuniens; *Rh*, nucleus rhomboidalis; *S*, stria medullaris; *Sm*, nucleus submedius; *SMA*, nucleus interstitialis supramamillaris; *SO*, nucleus supraopticus; *SOD*, nucleus supraopticus diffusus; *St*, stria terminalis; *STh*, nucleus subthalamicus; *Tr*, transition area; *VA*, nucleus ventralis pars anterior; *VM*, nucleus ventralis pars medialis, and *ZI*, zona incerta.

all the animals tested in the latter group responded violently to each shock. There were some exceptions. In several animals preoperative escape responses were transformed to rage responses after operation.

It was not possible to discern a good correlation between the intensity of the stimulus (as measured by separation of the primary and the secondary coil) and the type of response from one animal to another. Some cats responded to light stimuli. With others it was necessary to increase the intensity of current until the shoulder muscles contracted. Furthermore, there was no really satisfactory correlation between the preoperative and the postoperative stimulus thresholds. In general, the intensity was the same, but when changes were necessary, this change could not always be correlated with the type of behavior pattern which developed after operation. However, although these attempted objective tests were not altogether satisfactory from a quantitative standpoint, it is obvious that the animals which manifested definite changes in affective behavior responded to the shocks in a characteristically more violent fashion.

Responses to raucous, frightening noises were less constant, both before and after operation. While the noises caused a relatively large number of fear reactions, changes in response consistent with altered type of behavior were not general. This phase of the work may be dismissed with the comment that animals of all types were usually capable of showing true fear behavior in response to noise, but that this type of stimulation was frequently ignored entirely, both before and after operation.



Figs. 5-8.—Figure *5* (cat P16), right unilateral lesion in the mamillary region; *6* (cat P6), bilateral lesions in the anterior hypothalamic areas; *7* (cat P6), bilateral lesions in the dorsal hypothalamic nuclei, and *8* (cat P6), spots of gliosis in the extreme anterior tip of the ventromedial hypothalamic nuclei.

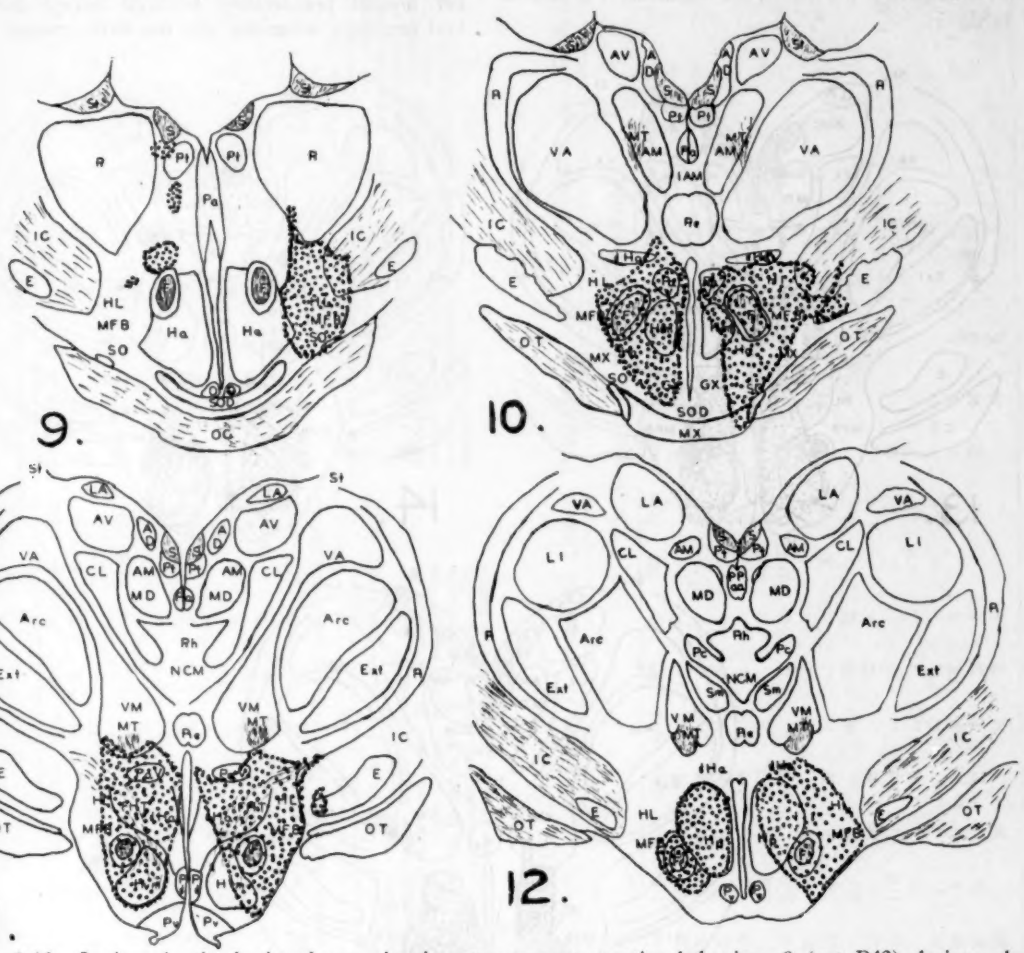
ANATOMIC OBSERVATIONS AND ILLUSTRATIVE PROTOCOLS

Because of the importance of a precise analysis of the destruction caused by the experimental lesions, the nuclear configuration correlated with

stereotaxic measurements as worked out by Ingram, Hannett and Ranson [35] was followed. The terminology used conforms with that adopted by the Association for Research in Nervous and Mental Disease (Rioch, Wislocki and O'Leary [36]).

The nuclei and well established tracts most frequently involved by the lesions are indicated in tables 1 to 4. Slight damage to one of these structures is indicated by *S*. *X* denotes complete destruction. The amount of damage between these two extremes was estimated in relation to the gross size of the remaining parts. *E* denotes a range of destruction from slight damage to loss of one-half the structure, and *M* indicates loss of more than one-half but less than complete destruction. These symbols used alone denote bilateral and symmetric involvement. Unilateral damage is indicated by *L* or *D* before the *S*, *E*, *M* or *X*; *L* means that the damage was restricted to the left side; *D*, to the right side.

The cats subjected to blank operations as controls showed no postoperative changes in behavior, and observations on them are not included in the tables. Sixteen cats remained friendly and displayed normal behavior despite the variety of destructive lesions indicated in table 1. The lesions in certain typical members of this group are illustrated in figures 1 to 8. Cat P6 (figs. 6, 7 and 8) showed no definite change in behavior despite destruction of the anterior hypothalamic area, Ganser's commissure, the suprachiasmatic nucleus and the supraoptic nucleus, with severe damage to the fornix, the perifornical area, the



Figs. 9-12.—Lesions in the brain of cats showing savage postoperative behavior. *9* (cat P42), lesions above the optic chiasm; *10* (cat P42), lesions in the region of the dorsomedial hypothalamic nuclei; *11* (cat P42), lesions in the region of the ventromedial hypothalamic nuclei; *12* (cat P42), lesions in the posterior hypothalamic area.

35. Ingram, W. R.; Hannett, F. I., and Ranson, S. W.: The Topography of Nuclei of the Diencephalon of the Cat, J. Comp. Neurol. **55**:333, 1932.

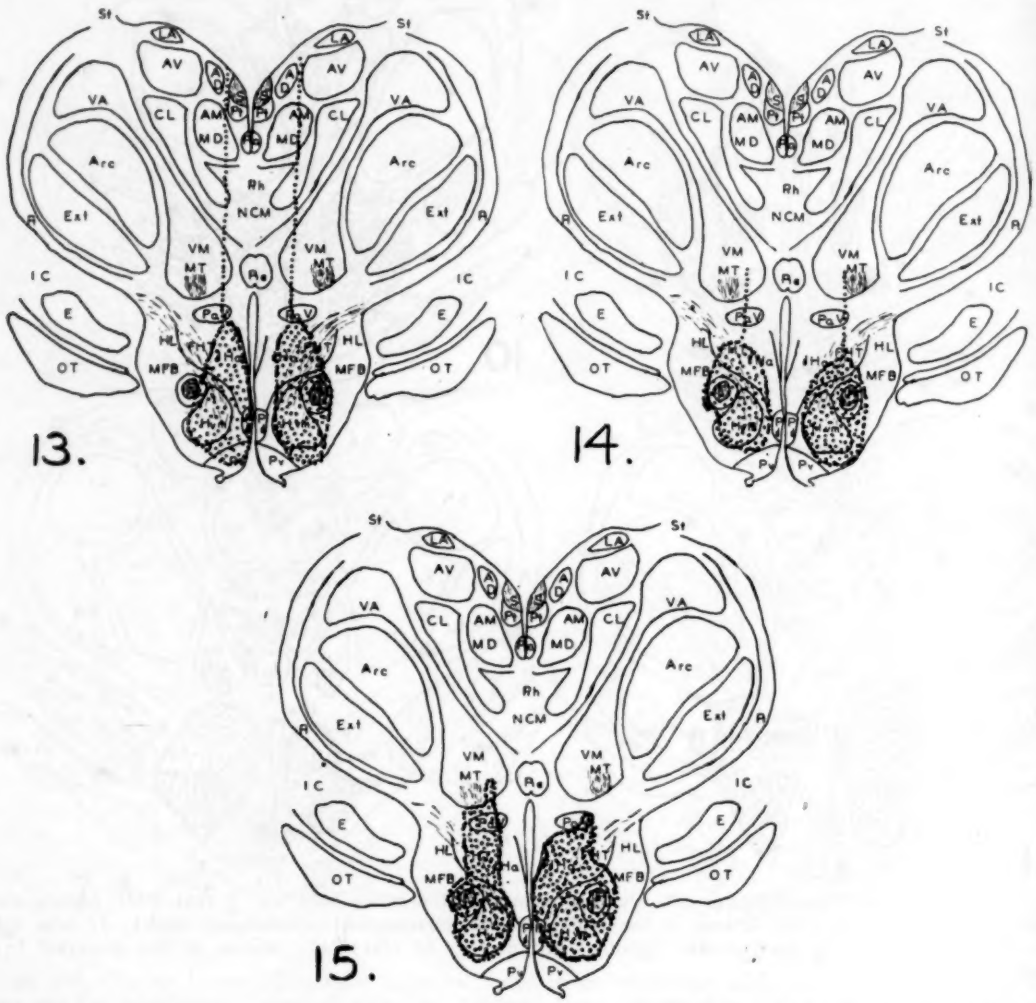
36. Rioch, D. M.; Wislocki, G. B., and O'Leary, J. L.: A Précis of Preoptic, Hypothalamic and Hypophysial Terminology, with Atlas, A. Research Nerv. & Ment. Dis., Proc. **20**:30, 1940.

nucleus supraopticus diffusus and the nucleus hypothalamicus dorsomedialis. Cat P16, which also showed no postoperative change in behavior, had a unilateral lesion on the right side extending from the stria medullaris down in a slanting direction through the hypothalamus to the right nucleus mamillaris lateralis. The following protocol is typical for the cats which showed no change in behavior. The results of electrical stimulation are indicated in columns A and B of table 1.

time making friendly advances to the observer. She was fond of her food but ate daintily. Her fur was spotless and well groomed. When in estrus she showed the usual female attitude toward male cats.

On April 28, she was operated on while under anesthesia induced with pentobarbital sodium, and bilateral lesions were placed in the hypothalamus with a direct current of 3 milliamperes. The time of electrolysis was thirty to sixty seconds. She was kept in a warm chamber (31 C.) during the period of recovery.

The period of convalescence was brief, and by May 3, she was eating all food. She showed no change from her normal preoperative behavior except during the first few days, when she was not lively enough to play.



Figs. 13-15.—Figure *13* (cat P20), bilateral lesions in the ventromedial hypothalamic nuclei; *14* (cat P29), bilateral lesions in the ventromedial hypothalamic nuclei, and *15* (cat P31), bilateral lesions in the ventromedial hypothalamic nuclei.

Cat P16.—This cat, a small but sexually mature female, was very friendly to people and to all other cats. When she was handled roughly, she did not use her claws or teeth in attempting to escape. She responded immediately to petting with such signs of feline pleasure as rubbing against the observer, purring and other vocalizations. She was exceedingly playful and when out of her cage would gambol with other cats or play with inanimate objects. She spent much

Even when sick she responded favorably to petting and did not become irritable when roughed. As shown in table 1, the incidence of rage responses when she was subjected to electric shock was not increased. The lesions are illustrated in figures 1, 2, 3, 4 and 5 and are summarized in table 1.

Table 2 summarizes the structures damaged or destroyed in the cats which showed slight

Table 1.—*Data on Sixteen Cats with Hypothalamic Lesions Displaying Normal Behavior*

TABLE 1.—*Data on Sixteen Cats with Hypothalamic Lesions Displaying Normal Behavior*

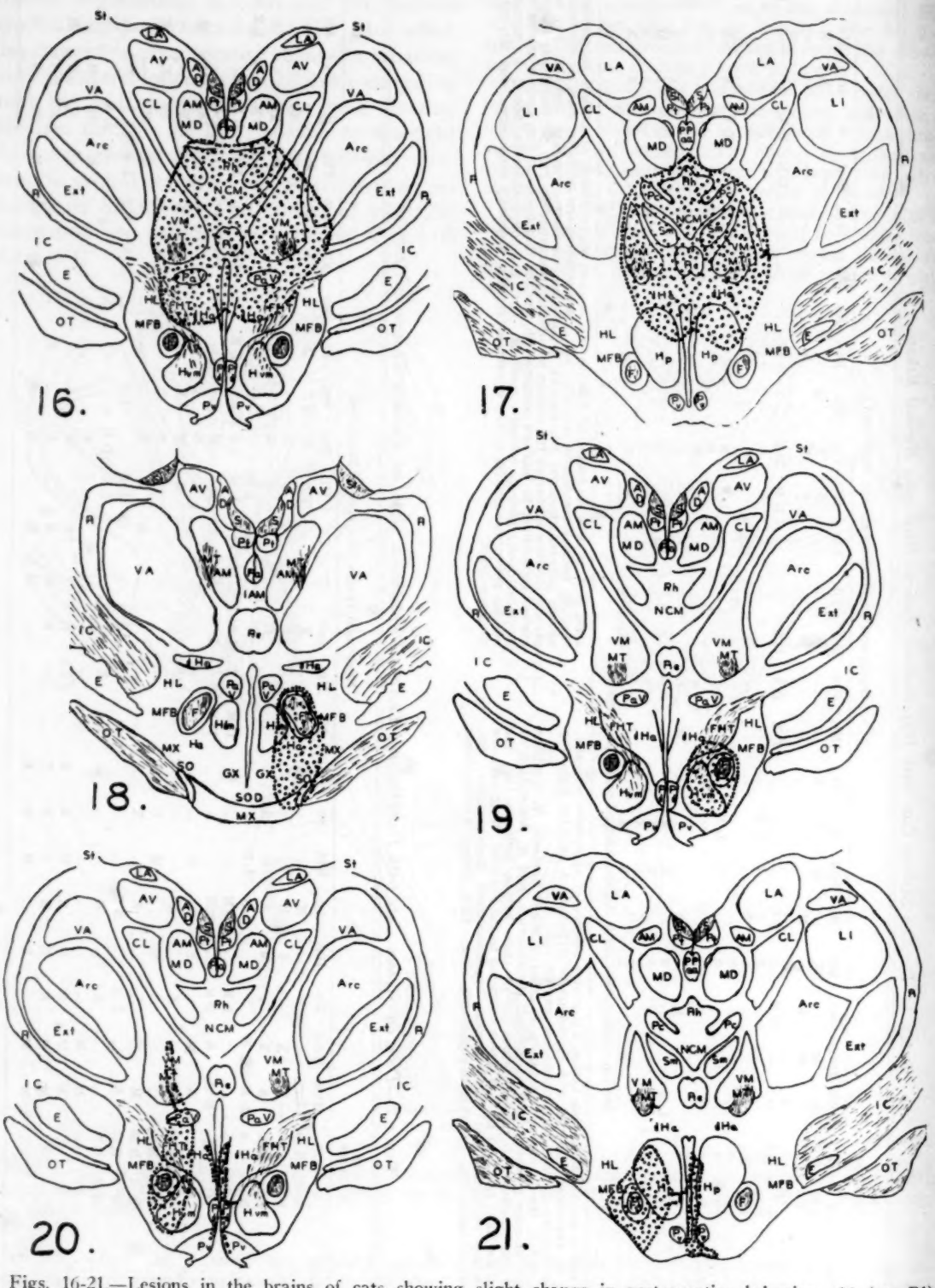
Cat	Behavior	Shock Response *		Nuclei and Tracts Damaged or Destroyed †																						
		B	A	Hvm	Hdm	Hp	Ha	dHa	Sod	So	O	Ml	Mm	Pav	Pd	Pv	Hl	Pvf	Sm	Re	Rh	Vm	Ncm	Fht	F	Gx	Mx	Mt
P 6	Normal	R 10	R 8	E	M	..	X	..	M	X	X	..	..	S	..	..	S	E	..	..	..	..	..	..	M	X	E	..
P11	Normal	0	0	..	M	E	..	M	..	..	..	..	..	M	..	..	S	E	..	S	..	..	..	E	..	..	..	X
P14	Normal	0	0	S	..	..	..	..	..	..	..	..	..	..	..	S	..	..	..	..	..	..	..	..	..	..	..	..
P16	Normal	R 8	R 4	..	..	D M	..	D E	..	..	..	D X	D S	D X	..	..	D M	D E	..	D M	..	..	..	D E	D X	..	..	D E
P17	Normal	E 13	0	..	..	..	S	..	..	L M	..	..	..	..	..	..	E	..	..	..	..	..	..	..	E	..	..	..
P19	Normal	E 1	R 3	S	..	..	..	S	..	..	..	..	..	S	..	..	..	..	..	..	..	..	..	..	..	..	..	..
P26	Normal	0	0	..	..	..	S	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
P32	Normal	0	0	..	L E	..	S	..	E	L M	X	..	..	..	..	..	L S	S	..	..	..	..	..	..	L X	X	X	..
P33	Normal	0	0	..	..	..	..	..	..	..	..	..	..	..	..	..	..	S	..	..	..	..	..	..	..	..	..	..
P39	Normal	R 1	0	..	..	..	E	..	..	..	..	..	S	..	..	..	..	M	..	..	..	..	..	..	..	..	..	X
P44	Normal	0	0	..	..	M	..	E	..	..	X	X	X	..	..	E	M	E	M	S	S	S	E	..	X	..	..	X
DI7	Normal	..	..	L M	..	..	S	..	E	X	..	..	..	..	E	X	..	E	..	..	..	..	..	..	..	X	X	..
DI11	Normal	..	..	L S	..	..	S	..	M	X	..	..	..	..	X	M	..	E	..	..	..	..	..	..	..	X	M	..
DI57	Normal	..	..	..	..	..	E	..	E	X	..	..	..	..	..	..	..	S	..	..	..	..	..	..	..	X	X	..
DI100	Normal	..	..	..	M	..	E	..	E	X	..	..	..	..	S	S	S	E	..	..	..	..	..	..	..	X	X	..
DI165	Normal	..	..	Section of stalk resulting in degeneration of supraoptic nucleus.																						

* In this table, and in the accompanying tables, 0 indicates absence of aggressive response; E, escape response; R, aggressive response, and C, convulsive response. A numeral following a letter indicates the number of times such a response was elicited.

† In this table, and in the accompanying tables, S indicates slight damage; X, complete destruction; E, slight damage to destruction of one-half the structure, and M, destruction of more than one-half the structure. D, placed before one of these letters, indicates that the lesion was limited to the right side, and L, that it was restricted to the left side. Hvm indicates nucleus hypothalamicus ventromedialis; Hdm, nucleus hypothalamicus dorsomedialis; Hp, tractus habenulopeduncularis; Ha, anterior hypothalamic area; dHa, dorsal hypothalamic area; Sod, nucleus supraopticus diffusus; So, nucleus supraopticus; O, nucleus suprachiasmaticus; Ml, nucleus mamillaris lateralis; Mm, nucleus mamillaris medialis; Pav, nucleus hypothalamicus paraventricularis; Pd, nucleus hypothalamicus periventricularis dorsalis; Pv, nucleus periventricularis arcuatus; Hl, nucleus hypothalamicus lateralis; Pvf, periventricular fibers; Sm, nucleus submedius; Re, nucleus reuniens; Rh, nucleus rhomboidalis; Vm, nucleus ventralis pars medialis; Ncm, nucleus centralis medialis; Fht, pallidohypothalamic fibers; F, fornix and nucleus periformicalis; Gx, Ganser's commissure; Mx, Meynert's commissure, and Mt, mamillothalamic tract.

TABLE 2.—*Data on Sixteen Cats with Hypothalamic Lesions Displaying a Slight or Variable Change in Behavior*

Cat	Behavior Change	Shock Response		Nuclei and Tracts Damaged or Destroyed																						
		B	A	Hvm	Hdm	Hp	Ha	dHa	Sod	So	O	Ml	Mm	Pav	Pd	Pv	Hl	Pvf	Sm	Re	Rh	Vm	Ncm	Fht	F	Gx	Mx	Mt
P 1	Slight	R 12	R 10	..	..	M	..	M	..	..	..	..	..	X	..	..	S	E	X	X	X	X	X	M	..	..	..	X
P 7	Slight	E 4	E 20	E	X	..	E	M	..	..	..	..	..	S	..	..	..	E	..	..	..	..	..	E	S	..	..	..
P 8	Slight	R 15	R 20	S	X	..	M	S	E	S	..	..	..	E	..	..	S	E	..	S	..	..	..	E	X	X	S	..
P15	Slight	R 3	R 6	L M	L E	..	..	L S	L E	..	..	..	..	..	..	..	L S	..	..	..	..	..	..	L X	L X	L X	L X	..
P27	Slight	0	R 3	..	..	L E	..	L E	..	..	..	L M	L E	L M	..	..	L M	S	..	L E	..	L E	..	L E	L X	..	..	L X
P37	Slight	C	C	D M	S	D E	..	D E	..	..	..	D X	..	D E	S	S	D E	E	..	..	..	D S	..	..	D X	..	..	D E
P38	Slight	C	C	S	M	..	E	..	E	..	..	..	..	..	..	..	..	S	..	..	..	..	..	..	S	M	M	..
P43	Slight	0	R 50	..	..	X	..	E	..	..	..	X	M	..	..	..	M	M	X	M	E	M	E	..	X	..	..	X
P10	Variable	R 14	R 50	..	..	..	M	..	M	E	X	..	..	..	..	..	..	E	..	..	..	..	..	..	S	X	E	..
P28	Variable	E 1	R 20	M	E	..	..	S	..	X	..	..	..	..	..	S	S	S	..	..	..	..	..	M	M	..	..	..
P36	Variable	R 6	R 22	S	..	L E	..	L E	..	..	..	..	..	S	..	..	L E	..	L S	..	..	L M	L S	L M	L S	..	..	L X
P40	Variable	R 16	R 24	..	..	E	..	E	..	..	..	L X	E	..	..	..	L E	D M	..	..	..	..	..	..	L X	..	..	X
DI31	Variable	..	..	M	M	..	..	..	..	..	..	..	..	..	..	..	..	E	..	..	..	..	..	M	M	M	..	..
DI73	Variable	..	..	E	M	S	M	E	M	X	X	..	..	E	X	E	..	M	..	..	..	..	..	..	X	M	M	..
DI86	Variable	..	..	M	M	E	E	E	E	X	X	..	..	E	M	S	..	E	..	E	..	..	..	..	E	..	..	..
DI136	Variable	..	..	M	M	..	E	..	M	X	E	..	..	..	M	E	..	E	..	..	..	..	..	M	M	X	E	..



Figs. 16-21.—Lesions in the brains of cats showing slight change in postoperative behavior. *16* (cat P1), bilateral lesions in the ventromedial region of the thalamus and the dorsomedial area of the hypothalamus above the ventromedial hypothalamic nuclei; *17* (cat P1), bilateral lesions destroying the ventromedial thalamic structure and the dorsomedial hypothalamic structures in the posterior hypothalamic areas; *18* (cat P15), bilateral unilateral lesion in the region of the left dorsomedial hypothalamic nucleus; *19* (cat P15), unilateral lesion in the region of the left ventromedial hypothalamic nucleus; *20* (cat P37), unilateral damage to the right ventromedial hypothalamic nucleus, and *21* (cat P37), lesions in the posterior hypothalamic areas.

postoperative changes in behavior and in those in which the behavior pattern was designated as variable. In these cats, as in the animals showing no change, a wide variety of hypothalamic structures was destroyed or damaged to some degree; yet no single structure was consistently injured or eliminated. It is perhaps significant, however, that the incidence of involvement of the regions of the ventromedial and dorsomedial nuclei was increased in these groups, as compared with the cats in the first group. It is also noteworthy that despite bilateral destruction of the ventromedial area of the dorsal thalamus in certain animals, these cats did not become truly savage. Cat P1 (figs. 16 and 17) is an example. This animal had an increased tendency to growl. When disturbed, he would use his claws in attempting to escape rough treatment but always responded favorably to petting and never displayed a vicious attitude. Interesting information on the relative absence of effect of a unilateral lesion is given by cat P15 (figs. 18 and 19) and cat P37 (figs. 20 and 21), which had extensive unilateral damage but did not become savage and might easily be classed as friendly. Cat P43 (table 2) is an interesting example of the effects of a lesion in the caudal part of the hypothalamus. This cat was somnolent for a week after the operation; yet he became irritated when roughed, growling and using his claws and teeth. He was not aggressive, however, and would respond immediately to petting with all the signs of pleasure of a friendly cat.

In tables 3 and 4 are tabulated the nuclei and tracts destroyed or damaged in the cats which became savage or showed pronounced change in the direction of savageness. Not one of these cats showed any postoperative tendency to respond favorably to petting. Their attitude and behavior have already been described, and this description holds for the group, with allowance for some individual differences with respect to certain items.

As the tables show, there was an extensive sampling of hypothalamic structures involved by lesions in these cats. It is important to point out that there was relatively little involvement of the dorsal thalamus, especially in the extremely savage cats. Also, while the lateral hypothalamic area was frequently encroached on, it was usually only slightly damaged, and in some cats it remained quite intact. The striking thing was the almost universal incidence of injury to the medial hypothalamic nuclei in these animals. In not all, however, was the degree of damage uniform. The most consistent results as to both incidence and degree were noted in the nucleus hypothalamicus ventromedialis. Most of the cats exhibiting a pronounced change in behavior had the ventromedial hypothalamic nucleus destroyed. All the savage cats showed complete destruction of this nucleus except cat P42 (figs. 9, 10, 11 and 12), in which the right nucleus was destroyed and approximately 60 per cent of the left nucleus was eliminated. The known lateral and dorsal fiber connections of the left nucleus, including the pallidohypothalamic fibers, were destroyed. It may, furthermore, be said that every cat in which the area of the ventromedial hypothalamic nucleus was destroyed became savage or showed a marked tendency toward savageness. In many of the cats with pronounced change in behavior which showed complete destruction of the ventromedial nucleus the only criterion which prevented classification of the behavior as savage was the fact that they would not attack inanimate objects. They would, however, savagely attack other cats or man. The true "savage" cats would attack anything when irritated. The animals of the group with pronounced change in behavior in which part of the nucleus hypothalamicus ventromedialis remained (cats P23, P35 and P41) were the least savage in this category; while they never responded favorably to petting, they frequently retreated threateningly instead of taking an aggressive attitude toward the observer. Not enough data were available on the behavior of cat DI102 to determine the exact degree of savageness it manifested.

In some of the savage animals the lesions were highly restricted and specific, as in cat P20 (fig. 13). Of course, when the nucleus hypothalamicus ventromedialis was destroyed, all the pallidohypothalamic fibers entering it were also eliminated or they were necessarily indicated as such in the table. Unless the nucleus was not completely destroyed, as in cat P42, the two structures must be considered as a unit. It is perhaps significant that in savage cat P42 the pallidohypothalamic fibers were eliminated, and in most of the cats showing pronounced change in behavior with incomplete destruction of the ventromedial nucleus this fiber system was severely damaged. A neighboring structure, the fornix column, was often affected in the cats with decided change in or savage behavior. However, in some cats it escaped, while in the animals showing normal behavior or slight changes in behavior it was sometimes destroyed. These observations tend to eliminate the fornix from consideration as a factor the loss of which contributes to "savageness." Figures 13 (cat P20), 14 (cat P29) and 15 (cat P31) illustrate hypothalamic lesions associated with savageness.

TABLE 3.—*Data on Seventeen Cats with Hypothalamic Lesions Displaying Decided Change in Behavior*

Cat	Behavior Change	Shock Response B	Shock Response A	Nuclei and Tracts Damaged or Destroyed: Hvm	Hdm	Hp	Ha	dHa	Sod	So	O	Ml	Mm	Pav	Pd	Pv	Hl	Pvf	Sm	Re	Rh	Vm	Nem	Fht	F	Gx	Mx	Mt
P 5	Decided	0	0	X	X	..	M	S	E	..	..	..	..	..	M	E	..	E	..	..	..	..	..	X	X	X	E	..
P12	Decided	E 4	R 40	X	X	M	S	E	..	..	..	..	..	E	M	E	E	E	..	..	..	..	..	X	X	M	..	S
P18	Decided	R 2	R 50	X	M	S	E	S	E	..	..	..	..	..	..	..	S	S	..	..	..	..	..	X	X	X	..	..
P23	Decided	C	C	M	S	..	S	..	S	..	..	..	..	..	..	X	..	..	..	..	..	..	..	..	..	X	..	..
P25	Decided	R 6	C	X	M	..	S	E	S	..	..	..	..	..	..	..	S	S	..	..	..	..	..	X	E	M	..	..
P35	Decided	0	..	M	M	M	S	M	..	..	..	L X	S	E	..	..	S	..	..	S	..	..	..	X	M	M	..	E
P41	Decided	R 1	R 1	E	..	E	S	S	..	..	..	..	..	..	..	..	E	..	..	..	..	..	..	M	M	..	S	..
DI 8	Decided	..	..	X	S	E	S	S	S	X	..	..	..	..	S	E	..	E	..	..	..	..	..	X	M	E	M	..
DI20	Decided	..	..	X	M	M	E	E	E	X	..	..	..	..	X	X	E	E	..	..	..	..	..	X	X	X	X	..
DI42	Decided	..	..	X	X	X	E	X	E	X	X	..	M	X	X	X	S	X	..	M	..	S	..	X	M	E	S	M
DI66	Decided	..	..	X	X	M	E	X	E	X	E	..	S	M	M	M	E	M	..	E	..	S	..	X	M	M	E	E
DI72	Decided	..	..	X	X	M	M	M	M	X	X	..	E	M	X	M	E	E	..	M	..	S	..	X	E	X	E	E
DI102	Decided	..	..	M	M	..	E	E	E	E	X	..	..	E	X	M	..	M	..	E	..	..	..	M	E	X	E	..
DI142	Decided	..	..	X	M	E	E	S	E	E	E	..	..	..	X	X	S	E	S	M	S	E	E	X	X	X	M	E
DI145	Decided	..	..	X	E	..	E	..	E	X	S	..	..	..	M	M	..	E	..	..	..	..	..	X	X	X	E	..
DI143	Decided	..	..	X	E	..	M	E	M	X	E	..	..	E	X	X	..	E	..	..	..	..	..	X	X	X	M	..
DI158	Decided	..	..	X	X	S	M	S	M	X	X	..	..	..	X	M	S	M	..	..	..	..	..	X	E	X	X	..

TABLE 4.—*Data on Nineteen Cats with Hypothalamic Lesions Displaying Savage Behavior*

Cat	Behavior	Shock Response B	Shock Response A	Nuclei and Tracts Damaged or Destroyed: Hvm	Hdm	Hp	Ha	dHa	Sod	So	O	Ml	Mm	Pav	Pd	Pv	Hl	Pvf	Sm	Re	Rh	Vm	Nem	Fht	F	Gx	Mx	Mt
P 2	Savage	E 5	R 50	X	X	E	E	E	E	L X	..	..	..	S	M	..	S	E	..	..	..	..	..	X	X	X	E	..
P 9	Savage	0	R 50	X	M	X	E	S	M	X	E	..	..	..	X	X	E	E	..	..	..	..	..	X	M	X	X	..
P20	Savage	R 1	R 50	X	E	S	..	E	..	..	..	..	..	S	..	S	S	..	..	..	..	..	..	X	S	..	..	..
P22	Savage	R 1	R 50	X	X	S	S	E	E	X	..	..	..	..	E	X	X	S	E	..	S	..	..	X	X	X	X	..
P24	Savage	C	C	X	M	S	M	..	M	..	X	..	..	..	X	X	S	E	..	..	..	..	..	X	E	X	M	..
P29	Savage	R 5	R 50	X	S	S	S	E	S	..	..	..	..	S	..	S	S	..	..	..	..	..	..	X	X	..	..	..
P30	Savage	C	..	X	L M	M	L E	M	S	..	..	..	..	M	M	M	E	E	..	..	..	..	..	X	X	E	E	D M
P31	Savage	..	..	X	E	..	S	E	S	..	..	..	..	E	..	..	S	S	..	..	..	..	..	X	X	M	..	..
P34	Savage	R 1	R 50	X	X	S	E	M	E	..	..	..	..	X	..	..	E	S	..	S	..	..	..	X	X	X	E	..
P42	Savage	R 1	0	M	M	M	S	M	S	..	..	..	..	M	..	..	E	S	..	..	..	..	..	X	X	X	X	..
DI1	Savage	..	..	X	E	E	M	E	E	X	S	..	..	S	X	M	..	E	..	..	..	..	..	X	X	X	X	..
DI30	Savage	..	..	X	X	M	E	X	E	E	X	..	M	M	M	M	M	M	E	M	E	E	..	X	X	M	..	M
DI61	Savage	..	..	X	X	M	M	M	M	..	X	..	E	X	X	X	E	M	..	S	..	..	S	X	X	X	X	E
DI135	Savage	..	..	X	E	..	E	S	E	X	X	..	..	..	X	M	..	E	..	..	..	..	..	X	X	X	X	..
DI141	Savage	..	..	X	M	E	E	S	E	X	E	..	..	..	X	M	..	E	..	..	..	..	..	X	X	X	M	..
DI154	Savage	..	..	X	S	S	S	E	..	..	..	..	S	..	..	..	S	E	..	..	..	..	..	X	X	S	..	X
DI155	Savage	..	..	X	S	S	..	E	X	X	..	..	..	..	X	M	S	E	..	..	..	..	..	X	..	..	..	..
DI157	Savage	..	..	X	S	S	E	E	E	X	..	..	..	..	X	X	S	E	..	..	..	..	..	X	E	X	X	..
DI87	Psychopathic	..	..	X	X	X	M	M	M	X	X	D X	M	E	X	X	M	X	..	..	..	..	..	X	X	X	X	D X

The following protocol is that of a typical savage cat:

CAT P20.—During the preoperative period, this cat, a mature, gray female, was very friendly although somewhat shy. She did not run after other cats and sought out the observer only when hungry; otherwise, she was content to stay in the background. When she was approached, she showed all the friendliness that cats can display. She was in estrus from April 16 to 20. During this time she sought out male cats in the usual manner and was not so shy with attendants. After this period she returned to her usual shy but friendly behavior. When aggressive cats attacked her, she cowered back, with ears flattened, but was never observed to make an aggressive move. When she was roughly handled, she tried to escape but did not use her claws and appeared bewildered. She did not play with other cats and only occasionally played with a ball or string. Her coat was well kept. She was slow and dainty in consuming her food.

On May 4, this cat was operated on while under pentobarbital anesthesia. She was then placed in a warm chamber (31 C.) for two days. On May 6 she licked her lips and growled when handled. She was placed in a leather harness to facilitate handling without danger to the observer; at this time she struck at the observer and was restless. On May 7 she ate her food. Before feeding she could be touched, but thirty minutes later she growled, hissed and struck with unsheathed claws when approached. On May 8 she assumed a typical "Hallowe'en stance," with ears flattened against the head, when any one approached her cage. She growled, hissed and struck if one attempted to touch her. She consumed all her food within fifteen minutes after it was placed in her cage. Before operation she often took several hours to finish her food. On May 9 she ate ravenously. Later she struck at the cage door when the observer approached her cage. Although she did not fly into a rage when food was placed in her cage, she could not be touched or petted. She showed marked horripilation and mydriasis when aroused and attacked anything which approached her. Usually when irritated she micturated several times and defecated until eventually nothing but mucus was passed. The volume of this mucus seemed abnormally large and may have indicated excessive secretion. Attempts to induce her to play with a ball or string brought on a fit of rage. When she was turned loose in a room with other cats, she attempted to retreat and remain in the background but attacked in a savage manner any cat that touched her. During the period from May 10 to September 21, this cat remained in a savage state. She was not observed in estrus during that time.[37]

On September 22, cat P20 was operated on while under anesthesia induced with pentobarbital, and all the neopallium anterior to the midregion of the posterior sigmoid gyrus was ablated on both sides.

37. Although no special effort was made to test the sexual behavior of the cats in this series, not one of the animals which showed notable change in behavior or became savage was observed to display the slightest interest in one of the opposite sex.

EFFECTS OF SECONDARY LESIONS AND ABLATIONS

Some clinical observers (Moniz and Lima [38]; Penfield and Evans [39]; Messimy,[40] and Freeman and Watts [41]) have reported the effects of frontal lobectomy and lobotomy on psychoses and neuroses, especially in persons suffering from manic or depressive states. According to the general theory on which such operations are based, these lesions eliminated malfunctioning areas and allowed other parts of the brain to recover their normal functions. Freeman and Watts [41] expressed the belief that ". . . prefrontal lobotomy acts upon the psychoses by removing more or less completely the emotional component with which the ideas are endowed . . ." and that this operation " . . . radically alters the personality of the individual in that it makes him no longer interested (affectively observant) in himself, either as an integrated collection of organs, or as a unit of society. . . ." Opinions vary as to the value of this procedure. The exact mechanism whereby ablation or lobotomy produces such changes is undetermined. Nevertheless, the frequency of occurrence of euphoria and loss of memory for recent events following these operations, as in Brickner's case (Brickner [42]), suggested the possibility that similar operations in the savage cats might alter their observed behavior. Jacobsen [43] and Jacobsen, Wolfe and Jackson [44] reported that, in addition to these two principal effects noted in man, monkeys and chimpanzees after frontal lobectomy no longer displayed the "temper tantrums" which difficult problems evoked in certain normal animals.

Removal of the frontal areas in cats has usually been observed to have little effect on emotional expression (Magoun and Ranson [26]; Barris [27]; Spiegel, Miller and Oppenheimer [25]).

38. Moniz, E., and Lima, A.: Symptômes du lobe préfrontal, Rev. neurol. **65**:582, 1936.

39. Penfield, W., and Evans, J. P.: The Frontal Lobe in Man: A Clinical Study of Maximum Removals, Brain **58**:115, 1935.

40. Messimy, R.: Les effets chez l'homme des lésions préfrontales, Ann. de méd. **45**:321, 1939.

41. Freeman, W., and Watts, J. W.: The Radical Treatment of the Psychoses and Neuroses, Dis. Nerv. System **3**:6, 1942.

42. Brickner, R. W.: Intellectual Functions of the Frontal Lobes: A Study Based upon Observations of a Man After Partial Bilateral Frontal Lobectomy, New York, The Macmillan Company, 1936.

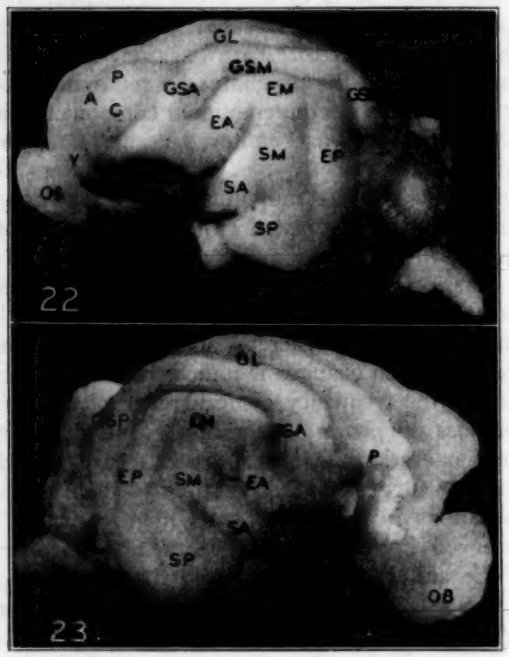
43. Jacobsen, C. F.: The Effects of Extirpations on Higher Brain Processes, Physiol. Rev. **19**:303, 1939.

44. Jacobsen, C. F.; Wolfe, J. B., and Jackson, T. A.: An Experimental Analysis of the Functions of the Frontal Association Areas in Primates, J. Nerv. & Ment. Dis. **82**:1, 1935.

Klüver and Bucy [45] found that in monkeys the removal of both temporal lobes, including the uncus and the greater part of the hippocampus, produced notable changes in emotional behavior in the sense that the motor and vocal reactions generally associated with anger and fear were not exhibited.

Various cortical areas were destroyed in some cats in this series of experiments to determine whether such lesions would be followed by additional alterations in affective behavior. Cat P20 was one of the animals treated in this manner, and the effect of lesions of the frontal lobe is illustrated by the continuation of the protocol begun in the preceding section.

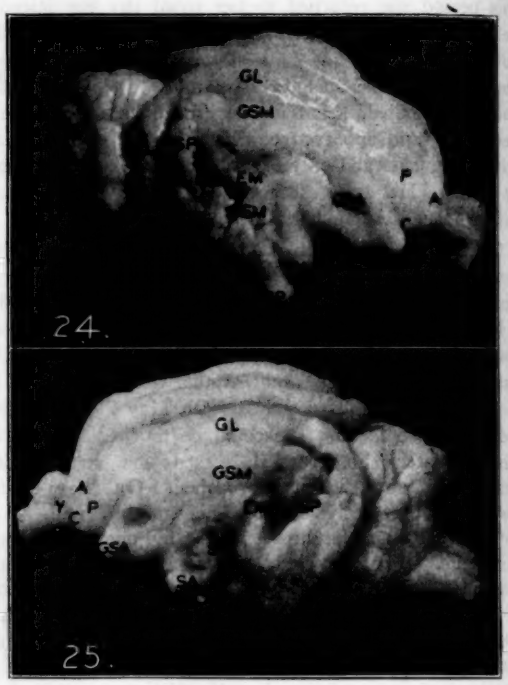
The convalescence was marked by ataxia, motor difficulty and loss of postural reactions. There were no definite cataleptic symptoms. Although the cat frequently slumped to the floor in an awkward position, with her legs sprawled under her body, she did not remain stationary in any fixed position while standing or when lying on her back in a trough. This cat progressively regained a fair degree of control of her muscles. She did not eat of her own volition for two weeks, and then would not eat when the observer was present. Before this she was fed by tube. On eight



Figs. 22-23.—*22*, photograph of the left side of the normal brain of a cat.

In this figure and in figures 22 to 29, the following abbreviations indicate the gross structures: *A*, gyrus sigmoides posterior; *C*, gyrus coronalis; *EA*, gyrus ectosylvius anterior; *EM*, gyrus ectosylvius medius; *EP*, gyrus ectosylvius posterior; *GL*, gyrus lateralis; *GSA*, gyrus suprasylvius anterior; *GSM*, gyrus suprasylvius medius; *GSP*, gyrus suprasylvius posterior; *H*, gyrus orbitalis; *OB*, olfactory bulb; *P*, gyrus sigmoides posterior; *SA*, gyrus sylvius anterior; *SM*, gyrus sylvius medius; *SP*, gyrus sylvius posterior; *T*, olfactory tract, and *Y*, gyrus proreus.

23, photograph of the right side of the brain of cat P20. In this cat the gyrus proreus was removed, and the following gyri were damaged: The right gyrus sigmoides anterior was removed, and the rostral half of the left gyrus was ablated, with extensive damage to the part remaining; the gyrus sigmoides posterior was damaged rostrally on both sides; there were slight damage bilaterally to the rostral portion of the genual gyrus, damage to both coronal gyri and slight damage to the rostral end of the left lateral gyrus. The left olfactory tract sustained a slight incision dorsally.



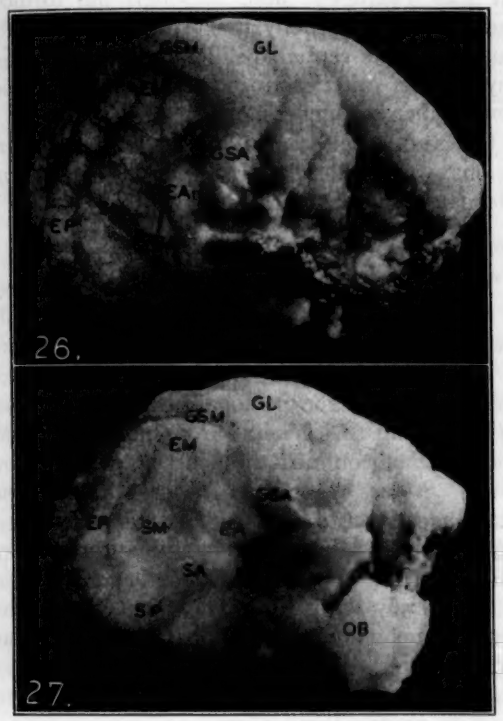
Figs. 24-25.—*24*, photograph of the right side of the brain of cat P31. In this cat, an attempt was made to damage the temporal region, in view of the observation by Klüver and Bucy [45] of a taming effect of such lesions in monkeys. The auditory projection area was therefore cauterized. As already mentioned, no change in emotional reactivity resulted in this experiment. On the right side, the anterior third of the cortex, the longitudinal part of the gyrus lateralis and the gyri adjacent to the inferior half of the lateral sulcus were undamaged. All other gyri on the lateral surface were damaged more or less, with the greatest destruction noticeable about 16 mm. dorsal to the ventral surface of the pyriform lobe.

25, photograph of the left side of the brain of cat P31. On the left side, all the gyri behind the posterior sigmoid gyrus showed varying degrees of damage or destruction. The area of greatest damage was in the gyrus ectosylvius medius. On both sides the damage affected not only auditory but visual areas, and sensory defects in these modalities were noticeable.

45. Klüver, H., and Bucy, P. C.: Preliminary Analysis of Functions of the Temporal Lobe in Monkeys, Arch. Neurol. & Psychiat. **42**:979 (Dec.) 1939.

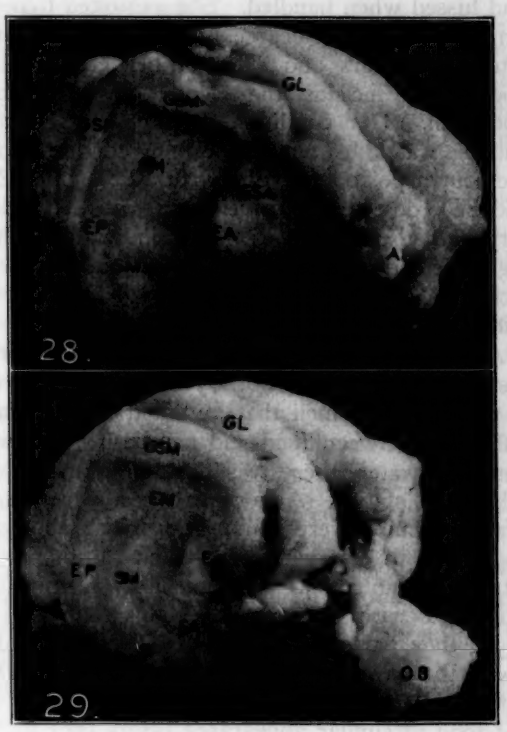
occasions she received subcutaneous injections of 100 cc. of sterile isotonic solution of sodium chloride. During these injections she struggled violently and for several minutes had good control of her facilities for attacking the observer, being able to strike repeatedly. In the absence of this intense stimulation, she would strike at the observer when approached, but could not unhook her claws for several seconds or until aroused to another effort. Despite this motor difficulty, there was no abatement of savageness, and she never responded favorably to petting; in fact, she growled and assumed an offensive position on the approach of a person or of other cats. On October 9 the cat became so enraged when handled that it was impossible to test her postural reactions. She urinated, defecated and showed all the behavior pattern of a savage cat. She was then killed; the head was perfused with a dilute neutral solution of formaldehyde, and the brain was removed for examination. Figure 23 is a photograph of the brain of this cat, showing the extent of damage and ablation.

Similarly, various portions of the cerebral hemispheres of cat P31 (figs. 24 and 25), cat P18 (fig. 26), cat P23 (fig. 27), cat P28 (fig. 28) and cat P25 (fig. 29) were extirpated or destroyed. Figure 22 is a photograph of a normal cat brain, shown for purposes of comparison. The degree of motor involvement varied from cat to cat. All the animals seemed to become exhausted after a relatively short period of intense activity. Some showed restlessness and hyperactivity. Cats P18 and P25 had a conspicuous tendency to be attracted by visual stimuli —following a moving object but wandering past or stopping when the object followed came to rest (Smith[46]). However, shortly after being



Figs. 26-27.—*26*, photograph of the right side of the brain of cat P18. In this cat, all structures anterior to the rostral end of the gyrus ectosylvius anterior were extirpated. The anterior surface of the remaining portions of the brain was necrotic and damaged.

27, photograph of the right side of the brain of cat P23. In this cat, all the neopallium rostral to the gyrus sylvius anterior was ablated or severely damaged. The olfactory bulbs appeared normal, but the superior surfaces of both olfactory tracts were damaged and eroded to a depth of approximately 2 mm.



Figs. 28-29.—*28*, photograph of the right side of the brain of cat P28. The right olfactory bulb was removed and the corresponding tract removed rostral to the olfactory tubercle, with involvement of the tubercle. Most of the olfactory bulb and tract on the left side were removed, but the lateral third did not show complete degeneration. The rostral end of the anterior sigmoid gyrus was damaged.

29, photograph of the right side of the brain of cat P25. In this cat, the gyrus proreus and the gyrus sigmoides anterior were ablated. The gyrus sigmoides posterior was completely extirpated on the right side, but a small degenerated portion remained on the left. Laterally, all the neopallium rostral to the gyrus ectosylvius anterior was removed. The rostral ends of the remaining portions of the neopallial gyri were damaged, and there were several pits. An area of softening in the region of the medial longitudinal fissure extended back into the genu of the corpus callosum. Both olfactory tracts were damaged posterior to the bulb.

46. Smith, K. U.: Hyperactivity in the Cat After Ablation of the Frontal Lobes and Its Relation to Visually Controlled Aspects of Behavior, Psychol. Bull. **39**:493, 1942.

handled, as for hypodermoclysis, they would rapidly chase the observer about the room and attack him when he stopped.

With 1 exception, cat P23 (fig. 27), these cats showed no definite change in emotional status. After the ablation of the neopallium anterior to the ansate sulcus, cat P23 did not show mydriasis, horripilation or the threatening retreat which she exhibited after the operation on the hypothalamus; however, she never responded favorably to petting and still growled and hissed when handled. She exhibited hyperactivity to a notable degree, bumping and pushing into the corners and sides of her cage. She opened the incision the second day after the operation. Her condition rapidly deteriorated, and she was killed when in a dying condition, on the fourth postoperative day. Cat P31 (figs. 24 and 25), despite extensive damage to the auditory and visual areas of the brain, with resulting deficiencies in hearing and vision, attacked viciously when touched and growled for several minutes after such a contact.

Although extensive damage to the olfactory system in cat P28 (fig. 28) caused such loss of the sense of smell that he could not tell the difference between food and a mixture of sawdust and water, there was no alteration in the behavioral status. A normal cat in which the olfactory bulbs were removed likewise showed no change in emotional expression. Both these cats when hungry would take bites of sawdust moistened with water in the same manner in which they partook of food. Neither cat swallowed the sawdust, and both soon spat it out, but they were unable to distinguish between it and food of similar appearance before they took portions of it into the mouth. Normal cats were always able to differentiate between the two; at least, they never tried to eat the sawdust mixture.

Several cats in the hypothalamic series had shown normal behavior, or only slight change, despite destruction of the fornices. The fact that Spiegel, Miller and Oppenheimer [25] observed pronounced rage reactions in cats after placement of lesions in the fornices, with involvement of the septum pellucidum anterior to the columns of the fornices, suggested the desirability of performing similar operations on cats which had shown a continuation of normal behavior, or only a slight change. Such lesions were placed in cat P32. No behavioral changes followed, although the fornices were extensively damaged and the septum pellucidum was largely destroyed just anterior to the columns of the fornices. A similar operation on cat P33 (with normal behavior) resulted in total destruction of the fornices, with extensive involvement of the septal structures of this area. Although this cat died on the second postoperative day, she was able to respond favorably to petting, and no behavioral change was noted. Also, cat P27 (with a slight change in behavior), after extensive destruction of the fornices with involvement of the septal area, did not show the slightest degree of change in the direction of savageness. If anything, she became less irritable and purred almost constantly when in the observer's presence.

Additional lesions were placed in this area in cat P29 (savage); and, although the fornices had been destroyed by the primary lesions, the damage to the septum pellucidum did not cause any further alteration in behavior. She lost none of her savageness.

Unless otherwise noted, all cats with secondary lesions were killed after a period of survival adequate for behavioral studies.

COMMENT

The correlation of the destruction of the area of the ventromedial hypothalamic nucleus with the development of a savage type of behavior is a new observation. Ingram [31] had observed such changes in the personalities of cats following lesions in this general area, but the destruction was not confined to such relatively narrow limits. Alpers [14] reported the case of a patient who was subject to periods of excitement bordering on rage. Microscopic examination revealed that most of the medial hypothalamic nuclei were severely damaged. There have been similar reports by other clinical observers, but pathologic lesions of the sort affecting the hypothalamus are rarely restricted enough for purposes of precise localization.

The destruction of thalamic structures or unilateral lesions in the hypothalamus in the present series did not result in the development of "savage" behavior. These observations differ from those of Kessler,[30] who described aggressive activity in cats following unilateral destruction of the thalamus or the hypothalamus. Kessler stated: "An unusual lability of the subject's emotional display . . ." resulted from unilateral diencephalic lesions "placed under more or less direct vision by following the stump of one optic nerve to the chiasmal region." His observations were not supported by detailed histologic study, and the placement of the lesions, according to his description, was vague.

Fulton and Ingraham [47] reported emotional disturbances following lesions placed in the base

47. Fulton, J. F., and Ingraham, F. D.: Emotional Disturbances Following Experimental Lesions of the Base of the Brain (Pre-Chiasmal), Am. J. Physiol. **90**:353, 1929.

of the brain rostral to the chiasm. This often cited observation was not supported by histologic delimitation of the structures involved. Spiegel, Miller and Oppenheimer [25] reported:

> . . . lesions encroaching upon the olfactory tubercles or isolated lesions of the tubercles were followed by distinct rage reactions (6 cases), particularly if the lesions extended medialward to the septum pellucidum.

Extreme rage reactions were obtained after transection of the fornices and damage to the septum pellucidum. These authors also observed somewhat paradoxic results after bilateral injury to the amygdaloid nuclei; both rage reactions and cataleptic symptoms followed such lesions. Their lesions were illustrated by gross cross sections, without apparent histologic analysis.

Microscopic scrutiny often reveals damage not apparent on gross examination. As an example, cat DI87 (table 4) had a section of the hypophysial stalk. This structure has no logical connection with behavior changes of the type dealt with here; nevertheless, the cat became savage, with additional psychopathic manifestations. Gross examination revealed no significant damage other than section of the stalk. However, microscopic study revealed an area of softening in the region of the tuber which destroyed the ventromedial nuclei and other structures. This injury was produced either inadvertently or from damage to vascular structures. Other cats subjected to section of the stalk retained a normal type of behavior. Cat DI165 (table 1) exemplified the usual result of such an operation, and microscopic examination of the brain of this cat revealed that only the supraoptic nuclei and the supraopticohypophysial tracts were degenerated.

While lesions destroying the region of the ventromedial hypothalamic nuclei are associated with the development of a savage type of behavior, one must be reserved in assigning to this specific cell group a function in the maintenance of normal, restrained, tame behavior. It must not be forgotten that this is an area of passage of fibers, and it is not impossible that there may be here a converging point for fibers which in other regions are scattered, so that relatively few are susceptible to destruction by restricted lesions. On the other hand, it is not impossible that in this area, in cats at least, important integrative mechanisms of an inhibitory nature exist.

The mechanism whereby the destruction of the region in question produces the loss of friendliness and the development of savageness is obscure. The fact that the condition persists long after convalescence and organization of the involved areas eliminates irritation as a causative agent. Evidence that electrical stimulation of this area produces euphoric or friendly behavior is almost nonexistent. It is perhaps significant in this respect that Gibbs and Gibbs,[48] in stimulating scattered areas throughout the brains of 400 cats, obtained purring in response to weak stimulation of the infundibular region in 3 animals. On the other hand, significant results of other sorts have been obtained with stimulation in hypothalamic areas other than the ventromedial nuclei. Ranson [29a] reported that he and his associates were successful in eliciting many of the manifestations of rage by stimulation of the region occupied by the medial forebrain bundle in the lateral part of the hypothalamus. Negative results were obtained by stimulation of the surrounding parts of the brain, such as the thalamus, the internal capsule, the septum, the infundibular stalk and the extrahypothalamic gray matter surrounding the fornix, or the fornix itself, except where this fiber bundle penetrated the lateral hypothalamic area. No rage effects were observed on stimulation of the medial portions of the hypothalamus. It should be added, however, that knowledge of hypothalamic function, as indicated by stimulation experiments, may be extended or modified as different technics are applied.

Rage reactions have been demonstrated by Bard and others after decortication and high decerebration. These responses have been explained on the basis of release phenomena. The "sham rage" exhibited by decorticate animals differs in certain respects from the rage reactions shown by the savage cats in the present study. Whereas the decorticate animals displayed these reactions only during the time in which outside stimuli were active, the cats in this series continued to respond for a considerable period after cessation of the stimulus. The attacks of the savage cats were well directed, unlike the blind fits of emotional display elicitable in the decorticate animals. For instance, after the destruction of the ventromedial region in cats P5 and D18, these animals would not attack inanimate objects, even avoiding a stick held by the observer in order to attack the latter's hand or arm. The emotional reactions of decorticate cats are not limited to displays of rage, for several investigators (Rioch [49]; Bard and Rioch [28]; Rioch and

48. Gibbs, E. L., and Gibbs, F. A.: A Purring Center in the Cat's Brain, J. Comp. Neurol. **64**:209, 1936.

49. Rioch, D. M.: Certain Aspects of the Behavior of Decorticate Cats, Psychiatry **1**:339, 1938.

Brenner [50]) have reported definite pleasure reactions in such animals. Bard (Bard and Rioch [23]) described a preparation in which not only the neocortex but the hippocampus and the pyriform lobe were removed on each side. This cat, in addition to purring, arched her back and made pleasure movements with her forepaws when suitably stimulated. The savage cats in the present series never responded with the slightest sign of pleasure; and although they might be touched with safety before feeding, they could not be trusted. They frequently attacked the observer at this time. Furthermore, while they ate avidly, they did not display the normal signs of enjoyment of their food.

Despite these differences in behavioral patterns, the responses of the two types of animals showed definite characteristics of emotional reactivity suggestive of rage. Normal emotion is undoubtedly impossible in both the "sham rage" animals and the "savage" animals. Any statement as to the relative importance of either the cortex or the ventromedial hypothalamic area with respect to each other and to the organism as a whole would be sheer speculation. If the hypothesis of "release" is accepted, it must be acknowledged that the structures inhibited have not yet been localized. Possibly they are located at several levels. The work of Bard [22] and that of Ranson [29a] suggest the possibility that at least part of the excitatory area may be situated in the hypothalamus. Lesions dorsolateral to the mamillary nuclei changed wild monkeys into tame ones. Bard expressed the opinion that the essential area was in the caudal part of the hypothalamus. Keller,[51] on the other hand, reported rage reactions after elimination of all brain tissue rostral to the midbrain. This observation makes necessary the investigation of excitatory areas other than those in the diencephalon. It will be recalled, however, that Bard denied that the picture of sham rage in its entirety can be obtained when the caudal part of the hypothalamus has been lost. The question of independent inhibitory activity by either the cortex or the ventromedial hypothalamic nucleus cannot be answered with certainty. Undoubtedly, the cortex inhibits the activities of subcortical structures, but the mechanism of this inhibition with respect to emotional behavior and the pathways through which it is accomplished have thus far only been postulated. Frontal areas have been extirpated without production of release phenomena (Magoun and Ranson [26]; compare Bellows and Van Wagenen [52]). Destruction of auditory areas of the cortex in monkeys resulted in suppression, rather than release, of rage phenomena (Klüver and Bucy [45]). Such operative intervention in the savage cats in this series caused no observable differences in the behavioral status.

It is questionable whether the lesions in the ventromedial hypothalamic nuclei interrupt inhibitory fibers from the cortex. The identity of such fibers in this region has not been properly established. Fibers of the periventricular system which connect the thalamus and the hypothalamus, and which are possibly a part of a corticohypothalamic system, are present here; but their activities have not been determined. There is no reliable anatomic evidence for concentration of such fibers in this particular area.

There is as yet inadequate information as to whether the destruction of afferent or efferent fibers related to this area has the same effect as destruction of the nuclei themselves. For instance, in cat P6, afferent fibers passing through the anterior hypothalamic area were destroyed without apparent effect. Also, in cat P1, the ventromedial portions of the thalamus were bilaterally involved in such a way that a great part of the thalamohypothalamic fiber system, as well as many pallidohypothalamic fibers, were destroyed without development of savage behavior. The only evidence on the positive side is suggested by the results in a single savage cat (P42), in which part of the ventromedial nucleus remained but in which the pallidohypothalamic and other dorsolateral connections were destroyed along the entire length of the nucleus. Further work on this score is indicated.

The part played by the area in question (whether the nucleus itself as a topographic entity or other elements located here) in relation to the activities of the nervous system as a whole in establishment of behavior patterns awaits analysis. Can it be that this area has an interlocking activity affecting the functions of the cortex? At any rate, present evidence indicates that in this restricted area in cats there is a nodal point at which the pathways mediating benign and malevolent behavior cross. When its activities are lost, behavior in cats reverts to an unrestrained savage, defensive and antisocial type.

50. Rioch, D. M., and Brenner, C.: Experiments on the Corpus Striatum and Rhinencephalon, J. Comp. Neurol. **68**:491, 1938.

51. Keller, A. D.: Autonomic Discharges Elicited by Physiological Stimuli in Midbrain Preparations, Am. J. Physiol. **100**:576, 1932.

52. Bellows, R. T., and Van Wagenen, W. P.: The Effect of Resection of the Olfactory, Gustatory and Trigeminal Nerves on Water Drinking in Dogs Without and With Diabetes Insipidus, Am. J. Physiol. **126**: 13, 1939.

The matter of corticohypothalamic relations remains a problem. Masserman [53] observed that cats were unable to adapt to direct stimulation of the hypothalamus in conditioning experiments and concluded that the hypothalamus may not be the "dynamic source or the seat of experience of affective states." Nevertheless, some experiments have indicated that destruction of the hypothalamus may alter the activity of the cortex. Grinker and Serota [8] noted temporary abolition of the cortical potentials following destruction of the hypothalamus in cats. Obrador Alcade [54] reported no return of cortical potentials following destruction of this area in acute cat preparations, and Kennard [55] observed that the bilateral destruction of the hypothalamus abolished or diminished the pattern of the cortical potentials in chronic monkey preparations. On the other hand, Morison, Finley and Lothrop [56] stated that in their experiments they found nothing to suggest an intimate relation between activity of the cortex and that of the hypothalamus. More work is necessary to place knowledge of corticohypothalamic relations on a sound basis.

A word must be said in regard to lesions involving olfactory structures. The part played by the olfactory sense in primitive behavior is well known. In my experience removal of the olfactory bulbs in normal cats and in cat P28 (with variable behavior) had no effect on emotional display. Similar results have been reported by other investigators (Spiegel, Miller and Oppenheimer [25]). On the other hand, Bellows and Van Wagenen [52] reported that the "believed" loss of olfactory sense due to injury of the olfactory tracts was associated with fear, rage or even maniacal behavior in dogs. It is not apparent that a careful histologic study was made, and it is important to note that in performance of the operations the frontal lobes were removed and various parts of the hypothalamic structures damaged. An account of the condition of the hypothalamus in these dogs would have been interesting. Although Spiegel, Miller and Oppenheimer [25] reported rage reactions following destruction of the fornices with involvement of the septum pellucidum in cats, the results of such lesions in the present experiments were negative. Dott and associates [11] noted no symptoms referable to section of the fornices and incision of the lower part of the septum pellucidum in their patients. Spiegel, Miller and Oppenheimer [25] reported rage reactions following bilateral lesions in the olfactory tubercles, especially when the lesions extended medially into the septum pellucidum. The mechanism of this effect needs further investigation.

A word should perhaps be added with regard to the avid appetite and tendency to gain in weight of many of the savage cats. My observations on the score of obesity were not sufficiently controlled with respect to variations in diet, metabolic factors and activity to contribute materially to the problems involved in the question of hypothalamic obesity. It is significant to record, however, that cats with hypothalamic lesions may become abnormally fat, an observation which adds the cat to the species in which the phenomenon has previously been noted. It is also of interest to note that Hetherington [57] stated the belief that obesity-producing lesions in rats are in the region of the ventromedial nucleus and that the lesions in the series of Brobeck and associates [34] were in the same vicinity. Further work on this score is indicated.

SUMMARY

The behavior of 42 selected, tame, friendly cats was studied for several weeks. In addition to observation of each animal's response to everyday environmental contacts, such as reactions to other cats, behavior toward the observer, feeding habits and cleanliness, the response to nociceptive electrical stimuli was noted. Bilateral lesions were then placed in the hypothalamus with the aid of the Horsley-Clarke stereotaxic instrument. Daily postoperative records of behavior were kept in a manner similar to that for the preoperative observations. Of the 42 cats, 13 remained friendly; 8 showed slight irritability; 7 displayed pronounced savageness; 10 became very savage, and 4 exhibited variable behavior in that their moods of friendliness alternated with periods of aggressiveness. Additional lesions were placed or ablations of various cortical regions were performed in 11 of these cats to determine whether such intervention would ameliorate savageness or produce rage reactions in the cats which had retained their normal friendliness.

53. Masserman, J. H.: Behavior and Neurosis, Chicago, University of Chicago Press, 1943.

54. Obrador Alcade, S.: Effect of Hypothalamic Lesions on Electrical Activity of Cerebral Cortex, J. Neurophysiol. **6**:81, 1943.

55. Kennard, M. A.: Electroencephalograms of Decorticate Monkeys, J. Neurophysiol. **6**:233, 1943; Effects on Electroencephalogram of Chronic Lesions of Basal Ganglia, Thalamus and Hypothalamus of Monkeys, ibid. **6**:405, 1943.

56. Morison, R. S.: Finley, K. H., and Lothrop, G. N.: Spontaneous Electrical Activity of Thalamus and Other Forebrain Structures, J. Neurophysiol. **6**:243, 1943.

57. Hetherington, A. W.: Non-Production of Hypothalamic Obesity in the Rat by Lesions Rostral or Dorsal to the Ventromedial Hypothalamic Nuclei, J. Comp. Neurol. **80**:33, 1944.

The brains were examined histologically. This series of brains was supplemented by specimens from another group of 28, the behavior of which had been noted after production of hypothalamic lesions for other purposes. Anatomic analysis showed that destruction of the region of the nucleus hypothalamicus ventromedialis was invariably associated with savage behavior of an extreme type, with complete loss of any friendly attitude. In many cases the lesions were highly restricted. Partial destruction of this nuclear area in many instances was associated with varying degrees of affective change. Evidence is not available to indicate whether the nucleus itself is the controlling factor or whether other factors participate.

CONCLUSIONS

Lesions destroying the immediate region of the ventromedial hypothalamic nuclei in cats result in loss of favorable response to friendly treatment and handling and in change from a friendly behavior pattern to one of malevolence and savageness, to a marked or an extreme degree. Attempts to produce this result by means of other lesions failed. Efforts to modify this behavior pattern by lesions or ablations of higher regions of the brain have also thus far essentially failed. The details of the mechanisms controlling or permitting the change are obscure.

Dr. W. R. Ingram advised in the carrying out of this experiment.

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MOTOR NERVE FUNCTION WITH LESIONS OF THE PERIPHERAL NERVES

A QUANTITATIVE STUDY

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In a great variety of abnormalities of the lower motor neuron it is of importance to determine the localization and the extent of the injury. It is also valuable to obtain a reliable and quantitative estimate of recovery of function or of the effect of treatment. A simple test has been devised which consists of application of a maximal electrical stimulus to the motor nerve and of recording, through the skin, the action potential of the muscle. An ordinary induction coil is suitable for stimulation, and the resulting electromyogram can be recorded on an electrocardiograph or any standard type of amplifier. Tests of this kind have been performed at an Army general hospital with which we are associated on patients with a large variety of peripheral nerve lesions during the past two years. In the present paper it is intended to describe the apparatus in detail and to point out its usefulness as an aid in the diagnosis and in observation of the progress of motor nerve function in these patients.

The present method employs a different approach to the problem than the electrical aids which have thus far been used in the diagnosis and prognosis of nerve injuries. The latter procedures may briefly be summarized as follows:

1. The classic method of determining the "reaction of degeneration" by faradic and galvanic stimulation. No accurate quantitative data are obtainable by this method, and in cases of partial lesions the results may be difficult to interpret.

2. Determinations of chronaxia and duration of strength. Significant results can be obtained only by plotting the entire curve for the duration of strength (Rushton [1]), while measurements of chronaxia give only one point on the curve and are of little value in making a quantitative estimate of muscle function.

3. The electromyographic method, in which spontaneous or voluntary activity is recorded. This procedure is of use when the nerve is inaccessible to stimulation. Records of this type, when used in conjunction with records obtained by the method described in this paper, frequently provide additional information of value. Details of these various methods can be found in papers by Bauwens [2]; Watkins,[3] and Schwab, Watkins and Brazier.[4]

MATERIAL AND METHODS

Recording.—The electrodes used in recording are silver or solder cups (5 to 10 mm. in diameter) with a small opening at the top sufficiently large to allow the insertion of an 18 gage needle. They are similar to the electrodes employed in electroencephalographic recording. As good contact of these electrodes is essential, the skin should be thoroughly cleansed with ether or alcohol. At the point of contact it is advisable to scarify the surface of the skin with a needle in order to release a small amount of tissue fluid. This procedure reduces the interelectrode resistance and minimizes the stimulus artefact. A drop of electrode paste is then placed in the hollow portion of the electrodes; they are brought into contact with the skin and fixed there by the application of a thin layer of collodion over their surface. If, during the course of the observation, the stimulus artefact tends to increase, it is generally an indication that drying has occurred at the point of contact of the electrode with the skin, resulting in increased interelectrode resistance. This can easily be remedied by applying a drop of saline solution through the small opening in the top of the electrode. Care should be taken not to allow the spread of the solution over the skin in the area of the recording or stimulating electrodes. This may result in large escapes of current and in stimulus artefacts.

The simplest method of recording is by means of an electrocardiograph or electroencephalograph. No special

1. Rushton, W. A. H.: Biol. Rev. **10**:1, 1935.

2. Bauwens, P.: Proc. Roy. Soc. Med. **34**:459, 1941.

3. Watkins, A. L.: Arch. Phys. Therapy **23**:76, 1942.

4. Schwab, R. S.; Watkins, A. L., and Brazier, M. A. B.: Quantitation of Muscular Function in Cases of Poliomyelitis and Other Motor Nerve Lesions, Arch. Neurol. & Psychiat. **50**:538 (Nov.) 1943.

shielding is required to take these tracings, and in general they can be obtained under all conditions in which electrocardiograms can be taken. If the potential can be suitably amplified and recorded on a cathode ray tube, as has been done in many of the experiments reported in this paper, more detailed observations as to its shape and duration can be made (fig. 2). The size of normal action potentials from muscles of the extremities is generally more than 5 microvolts (fig. 1). In most electrocardiographs the sensitivity can be sufficiently reduced to photograph such potential changes. It is necessary to calibrate the sensitivity of the recording instrument when records are made, so that the voltage of the potentials can be compared with those taken during subsequent periods of observation and alterations due to change in sensitivity of the recording apparatus recognized.

Stimulation.—The stimulating current for the motor nerve is applied by breaking the current flow in the primary of an induction coil with a Morse key or any suitable type of contact breaker. No appreciable stimulation occurs when the key in the primary circuit is closed, even when the subsequent "break" shock causes maximal stimulation of the motor nerve. The key in the primary circuit should be pressed down firmly and then released suddenly after the motor of the electrocardiograph is started.

In order to apply maximal stimuli to the motor nerves through the skin, high voltages may be required. These depend on the ratio of the windings of the primary coil to those of the secondary coil. Coils for strong stimulation generally have an iron core in the primary circuit. Strength of stimulus can always be varied by changing the relative position of the primary and the secondary coil to each other. The standard induction coils available in physiologic laboratories can be used for the purpose. Two stimulating electrodes lead from the secondary coil to the patient. One "indifferent" electrode consists generally of a large copper or zinc plate, about 3 by 5 cm., which makes contact with the patient's extremity about 10 to 20 cm. above the point along the nerve to be stimulated. The area of application should be rubbed with electrode paste, and a piece of cotton wool soaked in saline solution may be placed between the skin and the copper plate. To insure good contact, the electrode is fixed by a bandage or a rubber band.

The second stimulating electrode consists of a small round metal disk (1 cm. in diameter) attached to the end of a hardwood rod convenient for manipulation. Some gauze or linen may be tightly wound around the disk, in order to avoid any unpleasant burning sensation during stimulation. Before application of this localized stimulating electrode, the area of skin over the nerve should be cleaned and moistened with electrode paste and the cloth around the electrode soaked in saline solution. Stimulation itself does not cause pain, but the patient may be surprised by the mechanical response when the shock is first applied. He, therefore, should be told when to expect stimulation. During stimulation the extremity should be held in a relaxed, comfortable position. At the same time general movement should be prevented as far as possible, as excursions of the limb may result in the recording of movement potentials. As they always occur after the muscle action potential has been photographed, they do not interfere with the essential part of the recording, but they do cause alteration of the base line. A thin plaster shell provides a convenient support for the extremity from which the recordings are made. Several points along the limb can be lightly strapped to the support and excessive movement after stimulation easily avoided.

Points of Stimulation and Areas of Recording.—In the upper extremity all the main nerve trunks are accessible to stimulation, and records can be made from nearly all the superficial muscle groups. However, for the most part, muscles which have parallel fibers seem to be better suited than others, giving more synchronous and larger action potentials. Generally, one electrode is placed over the muscle under investigation and the other over an inactive neighboring area or over the tendinous portion of the muscle under study.

Points of Stimulation: In many instances the nerve to be stimulated can be located by palpation. The optimal location for the stimulating electrode varies from one subject to another and can be determined only by repeated shifting of the position of the electrode on the skin over the nerve until a maximal response is obtained.

Ulnar nerve: This nerve can generally be found without difficulty in the area above the medial epicondyle and is easily palpated.

Median nerve: This nerve runs in the depth of the elbow on the medial side, where it lies behind the lacertus fibrosus and is separated from the elbow joint by the brachialis muscle. In the forearm it passes between the two heads of the pronator teres muscle. The best point of stimulation is generally found to be proximal to the lacertus fibrosus, just medial to the tendon of the biceps muscle.

Radial nerve: This nerve can be palpated over 2 to 3 cm. of its length, as it winds posteriorly from the medial to the lateral side of the humerus, between the medial and the lateral head of the triceps muscle. Repeated trials are usually necessary, as it is often difficult to find the exact point at which maximal stimulation can be obtained.

Common peroneal nerve: In the lower extremity this nerve is the most readily accessible. It can be stimulated behind the head of the fibula at the lateral side of the popliteal space. The knee must be bent to a varying degree, depending on the position and tone of the tendon of the biceps muscle. The correct position is easily determined, and maximal stimulation is not difficult to obtain.

Tibial nerve: Maximal stimulation of this nerve is frequently difficult, and only a few recordings have been made from the muscles which it innervates.

Areas of Recording: Ulnar nerve: When the ulnar nerve is stimulated, recording from the muscles of the hypothenar eminence, the abductor pollicis muscle and part of the flexor pollicis brevis muscle is possible. The abductor digiti quinti muscle is preferable, as synchronous action potentials are usually obtained if one electrode is placed about 2 to 3 cm. distal to its point of origin and the other at the end of the muscle or on the skin over the first phalanx of the fifth finger.

Median nerve: Of the muscles supplied by this nerve, the flexor carpi radialis is most suitable, as it is superficially situated on the volar surface of the forearm. Eight to 10 cm. distal to its point of origin is the area in which the skin electrode is usually placed. Satisfactory records can also be made from the thenar eminence.

Radial nerve: Records are usually made from the brachioradialis and the extensor carpi radialis muscle when the radial nerve is stimulated. One electrode is generally placed several centimeters below the lateral epicondyle and the other in the region of the tendons of the extensors, at the wrist.

Peroneal nerve: In studies of this nerve, recording is done most frequently from the tibialis anterior muscle about 10 to 15 cm. below the patella and 1 cm. lateral to the edge of the tibia, but frequently more nearly synchronous and larger potentials are recorded about 3 cm. laterally, over the extensor digitorum longus and the peroneus longus muscle.

Satisfactory action potentials can be obtained from many regions other than those mentioned. The advisability of recording from a particular muscle frequently depends on the location of the nerve lesion and the set of nerve fibers involved. Thus, in certain cases of neuritis some muscles, although supplied by the same nerve, are more affected than others; and it is advisable to compare the potentials obtained from the various muscles innervated by the particular nerve under study. If small or complex potentials are obtained, better conditions for recording may be found after a slight shift of one electrode.

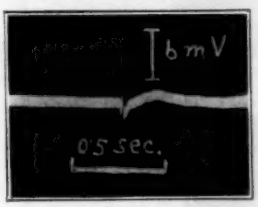

Fig. 1.—Action potential of the abductor digiti quinti muscle in response to maximal stimulation of the ulnar nerve.

Stimulation by induction coil, with recording on electrocardiograph.

During an actual experiment the following steps are taken in order to record the voltage of the muscle action potential in response to a maximal motor nerve stimulus:

The stimulating electrode is applied at various points along the course of the nerve. With practice, one can judge when the excitation of the nerve to a single stimulus is nearly maximal by observing the degree of movement in the contracting muscles and the deflection of the recording device. At this point, with the stimulating electrode held firmly in position, a record of the muscle action potential should be obtained. Without alteration in the position of the stimulating electrode, a second potential is then recorded after the strength of the nerve stimulus has been increased. If the first stimulus was maximal, the action potential in response to the second, and stronger, of the two stimuli will not have increased in size. Figure 1 shows a typical record obtained with an induction coil for the stimulation of the nerve and an electrocardiograph for the recording of the muscle action potential. This potential, of 6 microvolts, from the abductor digiti quinti muscle of a normal subject did not increase in size after a stronger stimulus was applied to the ulnar nerve.

RESULTS

If records of this type are taken under standard conditions, the potential in a normal muscle remains constant from day to day and over long periods (Harvey and Masland[5]). Comparison of the potentials from corresponding muscles on the two sides of the body rarely reveals a difference in the size of the potential of more than 15 per cent. Comparisons of this sort are indicated in the accompanying table in a variety of cases. It will be noted that although the potential values normally vary greatly for the different muscles, they are always within the same range on the two sides. In general, the voltage for any given muscle is within the same range, even in different persons. In the second part of the table the potentials recorded in a variety of clinical conditions are listed. Here, again, it is clear that the test is of value only when the normal and the abnormal muscle are compared or when the potential is recorded from the same muscle repeatedly over a considerable period.

Action Potential Voltages of a Number of Muscles in Response to Maximal Stimulation of the Motor Nerve

Muscle	Diagnosis	Action Potential, Microvolts	
		Right	Left
A. Normal Nerve			
Abductor digiti quinti.....	Normal	6	7
Adductor pollicis brevis....	Normal	7	....
Abductor pollicis brevis....	Normal	18	16
Flexor carpi radialis.....	Normal	15	13
Flexor carpi radialis.....	Normal	12	....
Brachioradialis.....	Normal	11.5	11.5
Tibialis anterior.....	Normal	12.8	10.8
Extensor digitorum longus (leg)	Normal	12.3	12.0
B. Unilateral Nerve Lesions			
Abductor digiti quinti.....	Peripheral neuritis (left)	5.2	2.4
Extensor digitorum communis	Hysterical wrist drop (left)	7.8	7.6
Brachioradialis.....	Traumatic neuritis (after recovery)	11.5	11.5
Brachioradialis.....	Traumatic neuritis	11.5	0.5
Tibialis anterior.....	Peripheral neuritis (right)	3.3	6.0
Tibialis anterior.....	Peripheral neuritis (left)	14.0	8.4
Tibialis anterior.....	Hysterical foot drop (left)	6.5	7.5
Extensor digitorum longus (leg)	Peripheral neuritis (left)	5.7	1.0

The following cases serve to illustrate the variety of problems in which a study of this type is useful.

Hysterical Paralysis.—During the past year numerous cases of localized weakness of one extremity or a portion of one extremity were encountered in this hospital. In many of these cases the loss of motor function was incomplete, and in the absence of sensory changes or alter-

5. Harvey, A. M., and Masland, R. L.: Bull. Johns Hopkins Hosp. **68**:81, 1941.

ation of reflexes, the differentiation of an organic and a hysterical abnormality was difficult. With a test available, such as the one described, partial loss of motor function can be detected, and the diagnosis may be made with more assurance.

A white man aged 26 was admitted with the complaint of weakness of the right hand, of sudden onset. There was no history of injury, and no obvious neuropsychiatric determinant was elicited.

The reflexes were normally active. Motor power was reduced in all the muscles of the hands to about one-half the normal, but the weakness was most noticeable in the extensors of the wrist and fingers.

The diagnosis on admission from the previous hospital was acute, peripheral, nonalcoholic neuritis of the right radial nerve, of undetermined cause.

Action potentials of the muscles supplied by the radial nerve were recorded in response to maximal electrical stimulation. The voltage of the potential from the right arm was the same as that obtained from the left (normal) arm, 6 microvolts (fig. 2*A*).

The patient was referred to the psychiatrist for examination, and a diagnosis of psychoneurosis, hysteria, in an emotionally unstable personality was made. He improved rapidly under treatment and was returned to duty.

A Negro aged 21, a sergeant, was admitted in December 1943 because of "paralysis" of the right foot, with foot drop, which had been present since May 1943, when he dropped an unfused bomb on his foot.

Examination revealed no power in the dorsiflexor of the foot and toes and great reduction in function of the plantar flexors. The reflexes were normal.

Electrical stimulation of the peroneal nerve resulted in dorsiflexion of the foot, and the resulting action potentials from the tibialis anterior and the extensor digitorum longus muscles were equal in voltage to those obtained from the corresponding muscles of the normal leg.

The psychiatric consultant made a diagnosis of psychoneurosis, hysteria. After the electrical stimulation he was able to move the foot more effectively but simultaneously began to complain vigorously of pains about the precordial region. After a few weeks in the hospital he was returned to duty.

In each of these cases, as a result of studies of the action potentials, a diagnosis of psychoneurosis, hysteria, could be made with more certainty than would have been possible on the basis of clinical observation alone. The use of electrical stimulation and visual observation of the mechanical response may be of help when the complaint is that of complete loss of movement, but when partial loss of function is present a definite conclusion cannot be reached with certainty without the simultaneous recording of the action potential in the affected muscle.

Progress of Recovery.—The fact that these potentials in response to maximal stimulation of the motor nerve normally remain constant from day to day makes the method useful in following the progress of recovery in nerve lesions.

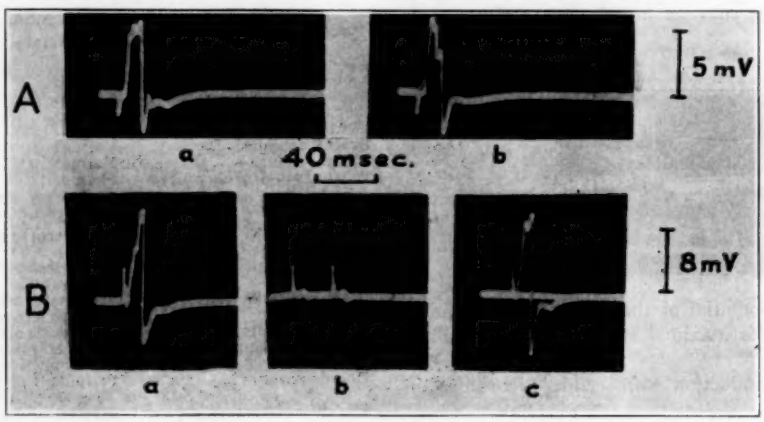

Fig. 2.—Muscle action potentials (cathode ray oscillograph) in response to maximal stimulation of the nerve (induction coil). (*A*) a case of hysterical paralysis: (*a*) normal side; (*b*) paralyzed side. (*B*) a case of traumatic neuritis of the radial nerve: (*a*) normal nerve (Nov. 1, 1943); (*b*) paralyzed nerve, two stimuli (November 1), and (*c*) paralyzed nerve (three weeks later).

Figure 3 illustrates the curve of recovery in a case of peripheral neuritis.

A young man, after several months of residence in the tropics, suddenly had paralysis of the peroneal nerve with foot drop. The action potentials from corresponding muscles of the normal and the abnormal leg were compared at frequent intervals. The potentials from the normal muscles varied from 15 to 16 microvolts. The curve shows several points of interest. The potential remained at about 50 per cent of normal for almost three weeks, during which there was no change in the degree of weakness on clinical examination. During the following three weeks there was a steady increase in the size of the potential, which appeared for a brief period before demonstrable clinical improvement. The curve then flattened out, with the potential remaining 3 to 4 microvolts below that on the normal side. A detailed study of peripheral neuritis will be presented later

Traumatic Neuritis.—Figure 2 *B* illustrates the results of study by this method in a case of neuritis due to pressure.

The patient awakened with complete paralysis of the radial nerve after sleeping on a train seat. Studies of the action potentials showed no function of the muscles in response to stimulation of the nerve. One week later improvement began, and the potential within three weeks became equal in size to that on the normal side. In this instance recovery was complete, in contrast to the result in the case illustrated in figure 3, and occurred much more rapidly.

In cases of temporary nerve paralysis of this type, as well as in those of partial nerve injury, in which it is desirable to know the exact degree of dysfunction, practical information can be obtained by the use of this method of study. Particularly in Army hospitals, a hysterical element may be superimposed in cases of partial nerve paralysis, and the extent to which this is present can be quickly ascertained if the action potential in response to maximal stimulation of the nerve is recorded.

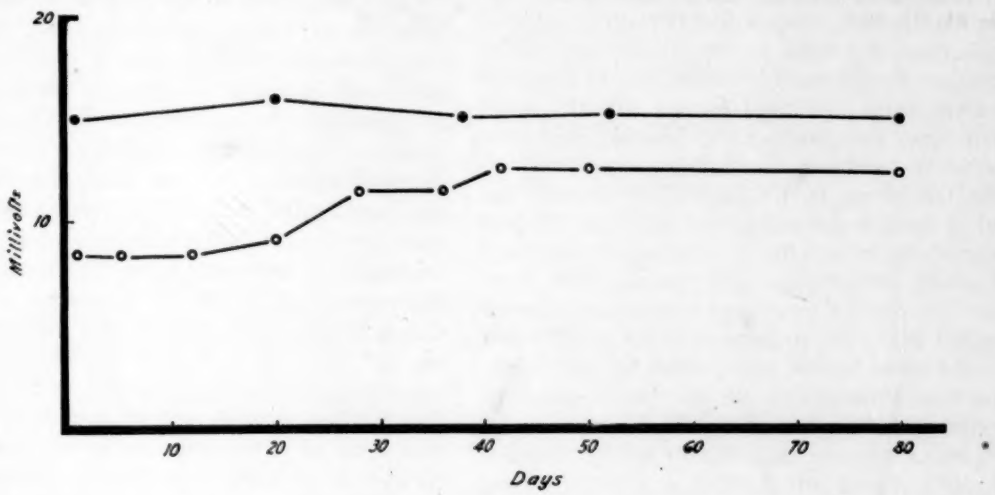

Fig. 3.—Recovery curve in a case of peripheral neuritis involving the peroneal nerve (line of hollow circles and dashes). The upper curve represents the variation in action potentials for the normal nerve.

COMMENT

Motor nerve function in various types of damage to the peripheral nerves, including peripheral neuritis, was investigated. The method which has been used represents a simplification of that described by Harvey and Masland [5] and offers many points of contrast to other types of electromyographic study. The following three points embodied in this type of study enable an accurate and objective estimation of nerve function: (1) When a maximal stimulus is applied to a motor nerve, the action potentials recorded under constant circumstances from the same muscle will show only slight variation over long periods; (2) a difference of more than 15 per cent is rarely observed when corresponding normal muscles of the extremities are compared; (3) whenever a portion of a motor nerve is the seat of an organic lesion, the muscle action potential is reduced, and the potential returns toward normal as the function of the nerve increases.

The records represent the response of the muscles to a maximal stimulus of the motor nerve fibers innervating it, in contrast to other types of records, in which potentials are produced by spontaneous or voluntary activity.

Studies of the type described appear to be of special advantage in the investigation of cases of partial nerve injury, peripheral neuritis and an exaggerated weakness of muscle power. The presence or absence of innervation can be ascertained, and repeated observations will reveal the progress of recovery. This is of special interest in the early stages following injury. During the first few weeks motor nerves which have not been permanently damaged will regain their function without undergoing degeneration. The recovery curve will then show an initial steep rise, as in the case of traumatic neuritis just cited. Further return of function will depend on the speed at which the damaged nerve fibers regenerate. Factors influencing this are the type of injury, its location and the nerve involved. As a rule, several weeks elapse during which no apparent progress results, as represented by the flat portion of the curve in figure 3.

In order to understand disorders of motor function, other types of study should be used in conjunction with the method described here and the results correlated. Spontaneous activity of single muscle fibers can be recorded with con-

centric needle electrodes (Adrian and Bronk[6]), while synchronous discharges of muscle groups are easily detected by the use of skin electrodes. An extensive study of various types of spontaneous activity has been made by Denny-Brown and Pennybacker.[7] Further, recording of potentials in response to voluntary motor activity may be the only method available when the motor nerves are not accessible to stimulation.

When the information from these two methods of study and the one described in this paper are correlated, a more complete picture of the motor abnormality can be obtained than by the use of any one of them alone.

6. Adrian, E. D., and Bronk, D. W.: J. Physiol. **67**:119, 1929.

7. Denny-Brown, D. E., and Pennybacker, J. B.: Brain **61**:311, 1938.

SUMMARY

The method described here allows an accurate and objective evaluation of muscle function in cases of lesions of the peripheral neuromuscular system. Records can be made from most of the muscles of the extremities.

The motor nerve is stimulated by an induction shock, and by records of the muscle action potential the localization and extent of partial lesions of the nerve can be established. An ordinary induction coil is sufficient for stimulation, and the muscle action potential can be recorded by means of an electrocardiograph or any standard amplifying system.

The extent of nerve injuries and the course of various types of lower motor neuron involvement can be determined. The method is also useful in the diagnosis of hysterical muscular dysfunction.

ELECTRIC SHOCK THERAPY

CLINICAL, BIOCHEMICAL AND MORPHOLOGIC STUDIES

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The purpose of this communication is to summarize our clinical observations and to describe more comprehensively studies of the blood and urine made immediately before treatments and at short intervals after convulsions.

CLINICAL OBSERVATIONS

Tolerance.—The following observations on the threshold for convulsions appear to be noteworthy: With 1 patient, three attempts made at five minute intervals to induce a convulsion by application of a current of 450, 500 and 550 milliamperes respectively for three-tenths second in the first treatment and two attempts by application of a current of 500 and 550 milliamperes for five-tenths second in the second treatment were followed by momentary loss of consciousness; five minutes later 600 milliamperes given for five-tenths second caused a severe convulsion, which lasted forty-five seconds. In the third treatment 650 and 675 milliamperes, again given at an interval of five minutes, induced only momentary loss of consciousness. In the fourth treatment 625 milliamperes given for five-tenths second induced a severe convulsion of forty-five seconds' duration. In the fifth and sixth treatments application of 600 milliamperes for five-tenths second also caused severe convulsions of fifty and forty seconds' duration respectively. In another patient 500 and 600 milliamperes given for three-tenths second evoked convulsions. In the third treatment four attempts with application of 600 milliamperes for three-tenths second were followed by four petit mal attacks. In the fourth treatment 600 milliamperes given for three-tenths second again caused a petit mal reaction; five minutes later only 300 milliamperes, but applied for five-tenths second, induced a major convulsion. In a third patient 450 milliamperes, given in two treatments for three-tenths and five-tenths second respectively, caused well pronounced convulsions; then two successive treatments with 450 milliamperes each, one treatment with 500 milliamperes and another with 550 milliamperes, each given for three-tenths second, were followed by petit mal attacks. Because the patient complained of nausea, no further attempt was made to induce a convulsion. In a fourth patient, in the first treatment, three attempts with 350 milliamperes and two attempts with 450 milliamperes, given for three-tenths and for five-tenths second respectively at intervals of five minutes, caused petit mal reactions. In the second treatment three attempts with 450, 550 and 600 milliamperes respectively for five-tenths second also produced only petit mal seizures; the fourth trial, with 600 milliamperes for five-tenths second, caused a fully developed convulsion of fifty seconds' duration. In a fifth patient 300 milliamperes given for three-tenths second produced a petit mal reaction. Then, three treatments with 350 milliamperes each for three-tenths second were followed by convulsions. In the fourth treatment three applications of 350, 400 and 450 milliamperes respectively for three-tenths second caused only petit mal attack, but in the fourth trial 450 milliamperes for three-tenths second produced a convulsion of sixty seconds' duration.

These cases illustrate our common observation that the patient's tolerance of the electric current may change at intervals of a few days or a few minutes. The tolerance may remain consistently high, though undergoing changes; it also may diminish considerably within a few minutes. To one of us (C.) atmospheric conditions had appeared to have a bearing on the dosage; on a dry, clear day a higher current was required than on a humid day. On the other hand, we also observed notable changes in the tolerance of the electric current in the same patient within a few minutes when atmospheric conditions appeared to be the same. Both change and consistency in the threshold for convulsions were observed with nearly equal frequencies throughout the treatments.

Reactions to the Electric Current.—A survey of the immediate reactions of our patients to the passage of the electric current revealed the fol-

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lowing facts: The latent period was commonly three to five seconds, and only occasionally twenty to thirty seconds. Petit mal reactions were observed with application of currents of from 350 to 675 milliamperes for three-tenths second. Their frequency was nearly the same with currents of from 350 to 450 milliamperes, 450 to 500 milliamperes and 500 to 550 milliamperes, and was notably less with currents of from 550 to 600 milliamperes. Severe and moderate convulsions were observed with a current of 300 to 600 milliamperes. However, with higher currents the frequency of severe convulsions greatly increased. With a current of 600 milliamperes given for three-tenths second the convulsions were severe in the large majority of treatments in the same and in different patients.

In some of the patients the reactions to the electric currents were unusual: One patient reacted to 550 milliamperes given for five-tenths second with the usual convulsion. He appeared relaxed for a minute or so; then he had another convulsion, though less severe and of shorter duration. With another patient the duration of the convulsion was remarkably long, namely, fifteen minutes with 500 milliamperes, three minutes with 600 milliamperes and six minutes with 300 milliamperes. One of our patients during the first three treatments reacted with jacksonian-like convulsions: After a latent period for about twenty-five seconds, the patient slowly turned his head to the left; his eyes and tongue deviated also to the left; then the fingers of both hands began to twitch; this was immediately followed by convulsive movements of the right side of the body and by a few clonic movements on the left side. After the convulsion there was a noticeable diminution in the muscular tonus on the left side as compared with that on the right side. In 1 patient a convulsion of fifty seconds' duration in the fourth treatment was followed by apnea for six minutes. With administration of nikethamide and artificial respiration regular breathing was reestablished.

Postconvulsive Reactions.—Nausea with or without vomiting was experienced by a few patients. Fear of the treatment was noted occasionally. One patient after the fifth treatment complained of being afraid of the treatments. Headache, from which he suffered after the preceding two treatments, may have been one of the reasons for his fear. Another patient, after a petit mal seizure, bade those around him good-by and said that he expected to die soon. Five minutes later he reacted with a convulsion to an increased dose; when he came out of the seizure, he complained of headache and expressed the belief that he was already dead. One of us (S.K.) and associates [1] had previously observed a similar reaction in a patient during metrazol treatment.

Complications. — Complications occurred in only a few of our 276 patients. Three had fractures of the head of the humerus. In 1 of them the fracture occurred during the third treatment. The patient felt greatly improved and was content to have his shoulder broken rather than to suffer the depression. Five patients had vertebral fractures. Curare was given prior to the treatment of 17 patients. One patient had fracture of the femur at the site of a former fracture; the first fracture had been followed by a severe depression.

THERAPEUTIC RESULTS

The therapeutic results obtained in 276 patients are summarized in table 1.

The relatively large number of patients in the schizophrenic group is due in part to the fact that it includes schizophrenic patients with prominent affective reactions—depression, tension and agitation—and with behavior and feeding problems. On the other hand, we were interested in trying out the treatment on schizophrenic patients without pronounced affective disturbance. Patients with essentially or prominently depressive reactions were included with the patients suffering from manic-depressive, involutional and undifferentiated psychoses, and a number of them, as just mentioned, were placed in the schizophrenic group. The relatively high rate of "recovery" and "improvement" among the last-mentioned patients is to be accounted for, largely, by the therapeutic results obtained in schizophrenic patients with depressive features. However, apparent "recovery" or "improvement" was obtained, also, in patients with fully

TABLE 1.—*Results of Electric Shock Therapy of Two Hundred and Seventy-Six Patients*

Diagnosis	Number of Patients	Patients Recovered	Patients Showing Improvement	Patients Showing No Improvement
Dementia precox (schizophrenia)	167	43	55	69
Manic-depressive psychosis, depressive type	60	32	16	12
Involutional psychosis (melancholia)	21	6	11	4
Undifferentiated psychoses (schizophrenic-affective features)	19	9	4	6
Psychoneurosis	9	0	5	4

1. Katzenelbogen, S.; Brody, M. W.; Hayman, M., and Margolin, E.: Metrazol Convulsions in Man, Am. J. Psychiat. **95**:1343, 1939.

developed schizophrenia, chiefly in the acute phase of their illness. Relapses among schizophrenic patients with depression were much more frequent than among essentially depressed patients. It is noteworthy that in some patients with a condition diagnosed as manic-depressive depression the treatment was followed by lifting of the depressive features and, as it were, by bringing to the fore of the schizophrenic reactions. It appears as though the latent schizophrenic condition was covered by the depression.

Of the patients who were difficult to manage, those with suicidal or homicidal tendencies and those dependent on tube feeding, the treatment was helpful to some, not only in ameliorating their behavior, but in leading to "recovery" and "improvement." Thus, of 28 of such patients, 12 "recovered" and 7 showed "improvement." [2] Some catatonic patients came out of their mutism and behaved as though recovered after only three treatments.

It is interesting to note that in general our patients showed improvement more frequently than not after only three or four treatments.

BIOCHEMICAL AND MORPHOLOGIC STUDIES

Specimens of urine and of blood from the arm vein were obtained before and after treatment from the patients, who were given no breakfast.

Urine.—The acid reaction of the urine remained unchanged after treatment in 38 of 45 instances. In 1 instance the reaction turned from neutral to acid after treatment; in 3 instances, from alkaline to acid, and in 3 instances, from acid to alkaline. In 46 instances the specific gravity showed significant changes after treatment. In 45 instances the reaction for sugar was negative within one hour and forty-five minutes after treatment. There was a trace of sugar before and none within one hour after treatment in 2 instances. In 43 instances the reaction for albumin was negative within one hour after treatment; there was a trace after treatment in 4 instances and a 2 plus reaction after treatment in 6 instances. Microscopic examination revealed nothing abnormal.

Blood.—The methods of analyses used in this study have been described in the articles cited.[3]

Changes in the constituents of the blood following convulsions are summarized in tables 2 and 3.

Both the cellular and the chemical constituents of the blood (amino acids, cholesterol, vitamin C, reducing substances, oxygen, total phosphorus, organic phosphorus, acid-soluble phosphorus, inorganic phosphorus, chlorides, sodium, potassium, calcium and magnesium), as well as the icteric index, showed changes after treatment; increases often being more frequent than decreases, except for the amino acids, which showed a decrease more than twice as frequently as an increase, and carbon dioxide, which was decreased after nearly all treatments.

TABLE 2.—*Sedimentation Rate and Morphologic Constituents of the Blood Which Showed Changes After Electrically Induced Convulsions* *

	Total No.	Treatments: Increase	Treatments: Decrease	Treatments: No Change
Red cells	41	27 (90,000 to 1,770,000)	13 (70,000 to 1,850,000)	1
White cells	41	25 (100 to 5,500)	16 (30 to 2,550)	..
Segmented neutrophils	31	14 (6 to 24%)	15 (4 to 24%)	2
Lymphocytes	29	14 (5 to 24%)	13 (6 to 20%)	2
Monocytes	26	15 (2 to 4%)	6 (1 to 4%)	5
Eosinophils	31	3 (1%)	5 (1%)	23
Sedimentation rate	33	20 (4 to 8 mm.)	12 (1 to 12 mm.)	1

* These changes appeared within one hour after convulsion.

COMMENT

Changes in many treatments were not great enough to be important in themselves; yet they gave significance to the more conspicuous alterations in that they emphasized a definite trend. Of the chemical constituents of the blood which undergo changes in the already old-fashioned insulin and metrazol therapies, the reducing substances, oxygen and carbon dioxide have been singled out as the most significant. The therapeutic effects of both these treatments have been attributed to anoxemia, induced by hypoglycemia in insulin therapy and, presumably, by lack of oxygen in metrazol therapy.[4] In convulsive

2. Baur, A. E., and Perrin, J.: Clinical Observations with Electroshock Therapy, Dis. Nerv. System. **5**:180, 1944.

3. Hawk, P. B., and Bergeim, O.: Practical Physiological Chemistry, ed. 11, Philadelphia, P. Blakiston's Son & Co., 1937. Myers, V. C.: Folin-Wu Method, in Practical Chemical Analysis of Blood, St. Louis, C. V. Mosby Company, 1924. Pijoan, M., and Walter, C. W.: A Micromethod for the Determination of Blood Cholesterol, J. Lab. & Clin. Med. **22**:968, 1937. Bessey, O. A.: Vitamin C: Methods of Assay and Dietary Sources, J. A. M. A. **111**:1290 (Oct. 1) 1938. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry: II. Methods, Baltimore, Williams & Wilkins Company, 1932. Fiske, C. H., and Subbarow, Y.: Colorimetric Determination of Phosphorus, J. Biol. Chem. **66**:375, 1925. Snyder, R., and Katzenelbogen, S.: The Distribution of Sodium, Potassium, Calcium, Magnesium, Inorganic Phosphorus, and Chlorides Between the Blood Serum and Cells of Normal Individuals, ibid. **143**:223, 1942.

4. Himwich, H. E., and Fazekas, T. F.: The Effect of Hypoglycemia on the Metabolism of the Brain, Endocrinology **21**:800, 1937. Himwich, H. E.; Bowman, K. M.; Wortis, J., and Fazekas, J. F.: Metabolism

(Footnote continued on next page)

therapy, it should be borne in mind that the deficiency of oxygen in the blood is limited to a short period of apnea during the tonic phase of the convulsions, and that in other phases there is hyperpnea, with corresponding increase of oxygen and decrease of carbon dioxide. But after the convulsion (especially within twenty minutes) in both metrazol and electric shock therapy, there is almost consistently an increase in the oxygen and reducing substances and a decrease in the carbon dioxide in the blood. Thus, if the therapeutic results in convulsive therapy are to be ascribed specifically to changes in the oxidative processes, it would seem appropriate to speak of hyperoxemia rather than of anoxemia.

In trying to understand the mode of action of shock therapies on the physiologic level, one should be cognizant of the fact that change in oxidative processes is not the only effect. Other metabolic alterations take place at the same time, as shown by previous morphologic and chemical studies [5] and by the present investigation. Prominent in the shock therapies considered in this study are the clinical reactions, the laboratory evidences of disturbance in the function of the vegetative organs and the metabolic changes. Thus, both clinical and laboratory observations are indicative of profound disturbances in the physiologic function of the person, in the vital organs and in the metabolism, which disturbances show a definite trend toward hyperactivity. Similar reactions of the person, of the organs and of the general metabolism are prominent in nonspecific protein therapy. It is not far fetched to postulate that the mode of action, namely, activation of the function of organs, attributed with good reason, I believe, to the nonspecific protein therapies [5] is equally applicable to the nonspecific shock therapies. But it should be added that the physiologic aspect is not alone to be considered. Multiple psychotherapeutic factors enter into the drama of any of the shock therapies, and contribute their share to the outcome.

TABLE 3.—*Chemical Constituents of the Blood Which Showed Changes After Electrically Induced Convulsions* *

			Treatments		
		Total No.	Increase	Decrease	No Change
Amino acids		50	16 (0.48 to 1.52 mg./100 cc.)	34 (0.49 to 3.19 mg./100 cc.)	..
Icteric index		19	9 (1.7 to 3.3 units)	..	10
Cholesterol		83	60 (8.2 to 91.2 mg./100 cc.)	23 (5.0 to 60.6 mg./100 cc.)	..
Vitamin C		74	57 (0.15 to 0.68 mg./100 cc.)	15 (0.1 to 0.23 mg./100 cc.)	2
Reducing substances		67	60 (11.0 to 72.3 mg./100 cc.)	2 (12.1 to 23.4 mg./100 cc.)	5
Oxygen		68	50 (4.13 to 12.28 vol. %)	16 (3.54 to 12.64 vol. %)	2
Carbon dioxide		67	3 (2.13 to 6.22 vol. %)	64 (5.29 to 33.68 vol. %)	..
Total phosphorus	Serum	19	15 (1.0 to 5.8 mg./100 cc.)	1 (0.3 to 12.1 mg./100 cc.)	3
	Cells	21	12 (0.8 to 6.8 mg./100 cc.)	8 (0.7 to 4.9 mg./100 cc.)	1
Organic phosphorus	Serum	18	12 (0.4 to 5.0 mg./100 cc.)	6 (0.2 to 9.0 mg./100 cc.)	..
	Cells	19	5 (0.4 to 11.6 mg./100 cc.)	14 (0.3 to 12.5 mg./100 cc.)	..
Acid-soluble phosphorus	Serum	20	16 (0.1 to 4.4 mg./100 cc.)	3 (0.1 to 2.8 mg./100 cc.)	1
	Cells	24	10 (0.6 to 3.0 mg./100 cc.)	13 (1.3 to 5.5 mg./100 cc.)	1
Inorganic phosphorus	Serum	52	45 (0.5 to 2.9 mg./100 cc.)	6 (0.1 to 1.3 mg./100 cc.)	1
	Cells	54	48 (1.2 to 8.3 mg./100 cc.)	6 (0.2 to 1.1 mg./100 cc.)	..
Chlorides	Serum	35	21 (5.7 to 19.9 mg./100 cc.)	12 (1.4 to 10.7 mg./100 cc.)	2
	Cells	36	34 (8.5 to 49.0 mg./100 cc.)	..	2
Sodium	Serum	92	67 (5.5 to 20.6 mg./100 cc.)	25 (3.2 to 27.6 mg./100 cc.)	..
	Cells	27	27 (4.2 to 25.6 mg./100 cc.)	..	..
Potassium	Serum	87	56 (5.7 to 22.8 mg./100 cc.)	30 (1.1 to 12.19 mg./100 cc.)	1
Magnesium	Serum	28	20 (0.2 to 1.2 mg./100 cc.)	8 (0.1 to 0.5 mg./100 cc.)	..
Calcium	Serum	41	27 (0.2 to 1.7 mg./100 cc.)	13 (0.1 to 1.1 mg./100 cc.)	1

* These changes were determined within eighty-six minutes after the convulsion.

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Case Reports

FAMILIAL INCIDENCE OF TUMORS OF THE BRAIN

Cerebellar Hemangioblastoma

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Many papers have been written concerning heredity as a factor in the development of tumors in the human body. Blank,[1] in 1941, reviewed the role of genetics in cancer research. Recently, we had occasion to treat the fourth member of a family to have a tumor of the brain. The occurrence of tumor of the brain in 4 members of one family strongly suggests a familial tendency in certain types of cerebral neoplasms. The predominating type of tumor identified in our cases was hemangioblastoma of the cerebellum. Cushing and Bailey,[2] in 1928, described at length many cases of this tumor. These growths were classified as simple cyst or gliomatous cyst until portions were removed for microscopic examination and a diagnosis of hemangioblastoma was made. The neoplasm occurs almost exclusively in the cerebellum, is usually cystic and has a familial tendency (Sargent and Greenfield[3]). Characteristically, but not always, the tumor is associated with angiomatosis of the retina, or von Hippel's disease.[4] Lindau,[5] in 1926, discovered that this lesion was frequently associated with angioma of the retina and cystic anomalies in other organs of the body. Gross examination may not reveal its presence, but necropsy may show small angiomatous nodules in the spinal cord, kidneys and adrenals, as well as hypernephroma and cysts or angiomas of the liver.

REPORT OF CASES

We present the cases of 4 members of one family with tumors of the brain. In 2 members—the father, aged 45, and a son, aged 23—tumors removed surgically were identified as true cerebellar hemangioblastomas. In the other 2 members, unfortunately, the type of the tumor was not verified. A daughter, aged 24, was thought to have died of a tumor of the brain, but operation was not completed.

The youngest son, aged 19, was the only member of the family who received treatment at this hospital. On the basis of the history previously obtained, that of the occurrence of cerebellar hemangioblastoma in 2 other members of the family, and of the neurologic signs, a diagnosis of cerebellar tumor, probably a hemangioblastoma, was made. Because of the poor condition of the patient and the tenseness of the dura in the posterior fossa, no attempt was made to remove the tumor. The patient died six hours after operation; permission for autopsy was not granted. Though the presence of a tumor was not verified. the neurologic signs and the presence of bilateral papilledema, with increased intracranial pressure, made likely the diagnosis of a cerebellar tumor. Careful examination of the retina revealed no angiomatous growth. The case records of the other 3 affected members of the family did not reveal the presence of angiomatous growths in any portion of the body. Since necropsy was not performed on any of the 3 patients who died, the presence of small angiomatous growths elsewhere in the body could not definitely be ruled out. Although we have no definite proof that members of this family all had Lindau's disease (combined hemangioma of the cerebellum and the retina), many features of the family history and of the neurologic findings speak for this condition.

Adequate information was obtained concerning the histories of the 3 members of the family who were treated elsewhere. These cases, and the case of the fourth member, are reported briefly.

CASE 1.—The father, aged 45, was first examined in August 1937, when a diagnosis of tumor of the cerebellum was made. On August 26 the presence of a tumor of the brain was verified at operation. A large cerebellar cyst and an associated tumor nodule, 1.5 cm. in diameter, were removed. Microscopic studies proved that the growth was a hemangioblastoma. The patient rallied for two days after operation; then sudden circulatory collapse developed, and he died within ten minutes. It was the opinion of the physician that death was due to coronary occlusion. There was no evidence of involvement of the retina. Autopsy was not performed.

CASE 2.—A daughter, aged 24, had died of a lesion which was probably a cerebellar tumor. Operation was not performed because the patient died suddenly. Autopsy was not performed.

CASE 3.—A son, aged 23, was examined at the University of Oklahoma Hospital in September 1943. Neurologic examination indicated a cerebellar tumor, and there was evidence of increased intracranial pressure. A tumor similar to that of the father was successfully removed. Microscopic studies revealed a hemangioblastoma. Convalescence was stormy, but the patient eventually recovered and is now without symp-

From the Neurological Service, Veterans Administration Facility, Hines, Ill.

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expression and control and in the sense of physical well-being constitutes a diagnostic triad which occurs more frequently than any single neurologic sign. They believe that the affective disturbances in mood and behavior fit in with the patient's previous personality makeup.

CHODOFF, Langley Field, Va.

WORK AND THE PLEASURE PRINCIPLE. IVES HENDRICK, Psychoanalyt. Quart. **12**:311, 1943.

Hendrick formulates the theory that work pleasure is not primarily displaced or sublimated sensual pleasure, but that it is the pleasure afforded by effective integration of the neuromuscular and intellectual functions. He calls the need of human beings for such pleasure the work principle, and he regards this principle as an expression of the instinct to master, the goal of which is control or alteration of environmental situations through the effective development of integrated intellectual and motor functions. The first objective evidence of the work principle is to be observed in the development of each partial function in infancy, when the stereotyped repetition of the function is replaced by the ability to modify the function effectively and to adapt it to tasks which satisfy the need to master. The work principle should therefore be regarded as evidence of maturity of ego function, and the repetition compulsion, as evidence that the ego is functioning inadequately for the skilful performance of a certain task.

PEARSON, Philadelphia.

CONCERNING THE PSYCHOGENESIS OF CONVULSIVE DISORDERS. LEO H. BARTEMEIER, Psychoanalyt. Quart. **12**:330, 1943.

Bartemeier believes that convulsions constitute a means of discharging destructive energy autoplastically. While all human beings have this innate preformed possibility of discharge, the predisposition of those who finally manifest a convulsive tendency consists in their predilection for the convulsive way of discharge. If the energy quantum is high enough and other ways are blocked, even the nonpredisposed person will turn to convulsions in order to relieve the mental apparatus of tension, and to a minor degree every person will show convulsive manifestations in one way or another.

PEARSON, Philadelphia.

FEAR OF DEATH. GREGORY ZILBOORG, Psychoanalyt. Quart. **12**:465, 1943.

Morale has to do with the avoidance of turning aggression against one's self and with the proper direction of aggression outward. The fundamental psychologic issue involved in the problem of morale is that of how one reacts to the fear of death, which is present constantly in mental functioning. It must be kept repressed, and in order to increase the repression one spends much time and energy in denying the possibility of death and in trying to master it. One's interest in news of violent deaths overlays an unconscious egocentricity. "It is not I who was killed."

When war comes to a civilized community, the fear of death, so well taken care of in peacetime, undergoes a crucial change. The civilian may still utilize the mechanism which makes him feel he is an exception and therefore cannot be killed. Not until members of his family and friends fall in battle does he come to grips with the fear of death by way of identification with those who were killed. Therefore compassion, an expression of the unconscious sense of guilt about those who have fallen, and scorn, a momentary sense of anger and hatred for the enemy who kills, become prominent components of the complex set of psychologic factors that group themselves around the repressed fear of death. Compassion and scorn become the core of morale, and the actual fear of death is mastered through hatred of the enemy. In the civilian who is exposed to bombing the fear of death is activated, but is countered by an increase in religious feeling and the mobilization of hatred. In this way the fear of death is transformed into a sadomasochistic combination, which on the socialized level is represented by awareness of grim hatred and of readiness to make great sacrifices to win the war. The accentuation of murderous drives is helpful in war, but if it occurred in times of peace it would lead to depressive states and to an increase in the rate of suicide. The rate of suicide is always higher in peacetime.

On the battlefield green troops become seasoned soldiers as they become angry, that is, as they convert their fear of death and hatred into hatred and aggression. In that fear of death which would be called neurotic, i. e., in the war neuroses, the victim hates, but he has a paralysis of motor aggression. He is unable to fight because he identifies himself with the dead as a result of a severe sense of guilt which antedates his military service. Those who are the most conspicuous misfits on the battlefront are the most ethically sensitive people, although they are socially the most useless.

Civilian morale might be improved if the general population were better informed about the losses and could see more of the wounded.

PEARSON, Philadelphia.

BODY AS PHALLUS: A CLINICO-ETYMOLOGICAL NOTE. HENRY ALDEN BUNKER, Psychoanalyt. Quart. **12**: 476, 1943.

The English word "body" is of unknown origin. Middle Gaelic, however, has the word bod, meaning penis. This corresponds to the psychoanalytic observation that body and penis are interchangeable in the unconscious. Bunker cites the fantasy of a 6 year old boy, which illustrates clearly that for this boy the word "body" meant penis. PEARSON, Philadelphia.

THE PSYCHOANALYTIC CONCEPT OF MEMORY AND ITS RELATION TO RECENT MEMORY THEORIES. ERNST LEWY and DAVID RAPAPORT, Psychoanalyt. Quart. **13**:16, 1944.

The psychoanalytic theory of memory is based on the view that memory traces are used by psychic forces, which find expression through them. The modes in which strivings make use of memory material vary significantly among such phenomena as everyday remembering, dreams and remembering of childhood experiences. The theories of memory deduced from experimental work by Lewin, Koffka and Bartlett show a striking parallel with the psychoanalytic theory.

PEARSON, Philadelphia.

HYPOGLYCEMIA AND TENSION-DEPRESSION. T. A. C. RENNIE and J. E. HOWARD, Psychosom. Med. **4**: 273 (July) 1942.

Rennie and Howard report on a series of 7 patients who had symptoms of a psychiatric disorder characterized chiefly by tension and colored by manifestations of depression. In addition, all the patients exhibited, as a conspicuous feature, a craving for food, which when satisfied resulted in temporary alleviation of the

emotional disturbances. Six of the patients were women, and 1 was a man. The hypoglycemic symptoms were in keeping with the flat dextrose tolerance curves obtained for all the patients. The hypoglycemia appeared to be secondary to the psychiatric disorder, since the former disappeared after the treatment of the latter. The authors emphasize that, since there are wide variations in the level of the blood sugar outside the accepted normal range, the dextrose tolerance test should be properly interpreted in terms of the individual personality. They conclude that when the specific complaints suggestive of hypoglycemia are presented, it is important to study the total personality and, in a certain group of patients, to treat the personality disturbances primarily.

SCHLEZINGER, Philadelphia.

PHYSIOLOGIC AND PSYCHOLOGIC STUDIES IN SPONTANEOUS HYPOGLYCEMIA. J. ROMANO and G. P. COON, Psychosom. Med. **4**:283 (July) 1942.

Romano and Coon describe the case of a married man aged 44 in which the original diagnosis of hysterical fugue was later proved incorrect and was replaced by a pathologically verified diagnosis of hypoglycemia due to a benign islet cell adenoma of the pancreas. The patient was an emotional, rigid, sensitive and dependent person who evidenced considerable repressed aggression. His illness was also characterized by recurrent episodes of confusion, uninhibited emotional behavior and disconnected movements. After the surgical removal of the adenoma the episodes were eliminated, but the essential nature of his personality remained unchanged.

The authors believe that the principal source of error in the diagnosis of a neurotic personality lies in the methods directed toward exclusion of positive data related to physical disease or psychosis. The need for the establishment of positive criteria of neurotic personality structure is emphasized.

In considering the relation between increased cerebral metabolism as a result of hypoglycemia and the resultant disturbances in intellectual, emotional and motor behavior, the authors suggest that inhibition and repression may be the physiologic and psychologic expressions of a basic phenomenon. The release of concrete intellectual activity, of less inhibited emotional expression and of less integrated neurologic behavior may result in all delirious patients as it did in this hypoglycemic patient. As in all deliriums, the primary psychologic disturbance is situated at the level of awareness. This disturbance of consciousness is more or less impersonal. On the other hand, the emotional behavior, which is dependent on the degree of disturbance of consciousness, is more individualized and specific in reflecting the personality structure of the delirious patient.

SCHLEZINGER, Philadelphia.

A PERSONALITY STUDY OF ALCOHOL ADDICTION. CHARLES C. HEWITT, Quart. J. Stud. on Alcohol **4**:368 (Dec.) 1943.

Hewitt employed the Minnesota multiphasic personality schedule and the Pressey senior classification test in an effort to determine the personality of persons addicted to alcohol. He claims that the schedule employed appears to be superior to any other inventory.

The case material consisted of 37 members of Alcoholics Anonymous, Inc., 15 other persons with alcoholism and 12 subjects who enjoyed the effects of alcohol but rarely drank in excess. The author concludes that addiction to alcohol seems to accompany, with few exceptions, deep personality disorders. Nearly all of the alcohol addicts, according to this study, exhibited a "marked psychopatic deviation which was often associated with neurotic, paranoid or schizoid trends."

GUTTMAN, New York.

PHYSICAL TREATMENT OF ACUTE PSYCHIATRIC STATES IN WAR. WILLIAM SARGANT, War Med. **4**:577 (Dec.) 1943.

Sargant points out that persons with reasonably good previous personalities do not generally break down or give up the fight against neurotic symptoms if their physical health is good. In treatment of acute conditions he emphasizes the need for sedation, preferably administration of the barbiturates intravenously or of paraldehyde by mouth. Forced feeding of enormous quantities of food and liquids is necessary with patients who have lost weight. This may be supplemented by small doses of insulin or by the Weir Mitchell technic. Sargant ends with the statement "Breakdown, even in the presence of great physical exhaustion and deterioration, is less common in an advancing triumphant army."

PEARSON, Philadelphia.

MANUAL OF EMERGENCY TREATMENT FOR ACUTE WAR NEUROSES. LAWRENCE S. KUBIE, War Med. **4**: 582 (Dec.) 1943.

Most war neuroses begin as acute disorders. These disturbances frequently develop gradually; i. e., the collapse occurs only after cumulative stress. Paramount in this stress are the effects of fatigue and hunger, of incessant and increasing danger and of a series of repeated, rapidly successive, narrow escapes. It takes time to recover from the effects of sudden danger; but when one threat is followed at once by another, there is no interval in which the emotional reverberations can die out, and the person spends his days and nights living as though he were engaged in an incessant struggle to escape from or master the original danger. Each successive escape contributes to the secretly mounting tension. At some point the tension becomes manifest, usually the first sign being a specific disturbance of sleep and terror dreams. Once sleep is disturbed, fatigue increases rapidly, giving rise to moments in the waking hours in which the patient lapses into momentary half-dissociated states. At such moments sudden movements, noises or lights cause a startled panic. As the fatigue and moments of terror spread through the day, the patient becomes unstable and depressed and feels alienated from his comrades. There are also many subtle changes in his daily behavior.

As any man may break under sufficient stress, prevention depends on the recognition of incipient stages and on steps which can be taken to stave them off. There are several important preventive measures: 1. Officers and men should be taught to recognize early any disturbances of sleep. 2. As soon as sleep disturbances are noted, sedation, with a combination of two sedatives, should be given; one of the sedatives should act rapidly, and the other should act slowly and have a sustained action. 3. A low light should be kept burning in the sleeping quarters throughout the night. 4. The soldier should be awakened swiftly and be given a cold drink or black coffee, followed by amphetamine sulfate, as soon as he is awake. 5. Rest periods during the day should not be silent, but the soldier should be kept amused and interested.

After the neurosis is established, the emergency treatment is as follows: (1) Controlled sleep, physical restoration and friendly group companionship; (2) superficial catharsis through companionship in isolation, "gentling" and opportunity for cathartic release; (3) prolonged sleep under continuous narcosis; (4) group methods of psychotherapy; (5) deconditioning procedures; (6) hyponarcoanalysis and the induced hypnagogic reverie, and (7) in cases in which the patient is inaccessible to any contact, even under narcosis, one or two electric shock treatments or sustained periods of hypoglycemia, without coma or convulsions.

PEARSON, Philadelphia.

PSYCHOSES IN OFFICERS IN WORLD WAR II. ADDISON M. DUVAL, War Med. **5**:1 (Jan.) 1944.

Duval reports on the study of 100 officers admitted consecutively to St. Elizabeths Hospital and compares this group with a somewhat similar group of officer patients in World War I.

Twenty-seven patients gave a history of previous mental illness, and 10 had previously been treated in hospitals for mental disease. At least 20 of the 27 patients would have been rejected on induction if they had been given an adequate psychiatric examination. The ages of the entire group varied from 20 to 49 years. Eleven men had service of less than one month, and 45, of less than one year. In many instances situational factors were important in producing the psychotic attack. Thirty-four patients had manic-depressive psychoses, and 30, schizophrenia. With 10 patients electric shock gave good results. Seventy-four patients have been discharged from the hospital, 49 as recovered or socially recovered, 20 as showing improvement, 2 as showing no improvement and 3 as free from psychosis.

PEARSON, Philadelphia.

HYSTERICAL VISUAL DEFECTS. PAUL T. MCALPINE, War Med. **5**:129 (March) 1944.

McAlpine reports briefly 9 cases of amblyopia of hysterical origin and 1 case of amblyopia regarded as a symptom of dementia precox. Hysterical amblyopia is of infrequent occurrence and must be differentiated from retrobulbar neuritis, toxic neuritis and amblyopia due to disease of the central nervous system.

PEARSON, Philadelphia.

RETROGRADE AMNESIA. W. MAYER-GROSS, Lancet **2**: 603 (Nov. 13) 1943.

Mayer-Gross tested retrograde amnesia in 102 trials on 46 patients. The subjects were all psychotic, without impairment of memory; the author excluded patients whose full cooperation was doubtful. Tests were made for memory of a series of four picture cards the minute prior to the administration of electric shock therapy. After twenty-four hours or less the patient was tested for memory recall and for recognition. Only 2 patients recalled all cards, and of all 102 tests only 29 resulted in complete recognition. The author stated that this result indicates that retrograde amnesia is not a universal symptom after epileptiform convulsions. There was a direct relation between the time the pictures were shown and recall and recognition; the pictures shown first were better remembered than those shown next, and the pictures shown last were least often recalled or recognized. Thus the defect was true retrograde amnesia. The results of testing three to eight hours after the shock were compared with those of tests made twenty-four hours later. The first picture shown was consistently better remembered a few hours after the seizure than a full day later. The author concluded that this observation indicated that "it is not a matter of indifference at which time the patient is tested for retrograde amnesia." In only one tenth of the experiments did the retrograde amnesia last longer than one minute.

Mayer-Gross discusses the theories explaining this phenomenon. The most popular current theory among neurologists and psychiatrists is that the traces, or engrams, of what has gone before the accident are destroyed. The author believes that the difference between recall and recognition in his tests is too great to support this theory by which he means that one could not recognize so well what one could not recall if the subject matter had been actually destroyed or blotted out. Rather, he prefers the theory that the consolidation of recent memories is interrupted; there is a retroactive inhibition similar to that known to normal psychology.

He cannot explain the individual differences in retrograde amnesia found among his subjects. In patients with head injuries the variations might be explained by differences in local cerebral damage, but the duration of electric shock and its clinical symptoms vary so little from subject to subject "that one is forced to surmise the presence of an unknown personal factor." Nor can he explain the clinical fact that patients in post-traumatic drowsiness will describe an accident in detail which they cannot remember when completely conscious later. This phenomenon seems to parallel the fuller recollection of the first picture in the early test of his series.

MCCARTER, Philadelphia.

Diseases of the Brain

INCIDENCE OF THE CHANGES IN THE RETINAL VEINS IN MULTIPLE SCLEROSIS. J. V. TRUESCH and C. W. RUCKER, Proc. Staff Meet., Mayo Clin. **19**:253 (May 17) 1944.

Rucker describes perivenous sheating in the retinal veins in cases of multiple sclerosis and asserted that in most of the cases in which this sign was present a definite or a presumptive diagnosis of multiple sclerosis had been made.

Truesch and Rucker report on the examinations of the fundus in 52 patients with multiple sclerosis, in all of whom careful search was made for perivenous sheathing. All the examinations were made through dilated pupils; all observations were confirmed, and all borderline cases were discarded. Of the 52 patients studied, 10 (19 per cent) showed the changes described as perivenous sheathing. The ages of the 10 patients varied from 18 to 51, 5 being women and 5 men. The average time of appearance of the sheathing after the first symptom of multiple sclerosis was four and one-third years. The condition is not related to the occurrence of pallor of the optic disks nor to a history of previous blurring of vision.

ALPERS, Philadelphia.

LEBER'S DISEASE: REPORT OF FOUR CASES IN ONE FAMILY. C. S. ALEXANDER, Texas State J. Med. **39**:301 (Sept.) 1943.

Leber's disease is defined as hereditary bilateral primary optic nerve atrophy. It is transmitted almost entirely by the female, who is usually unaffected, although it may rarely be transmitted by the male directly or indirectly through the daughters. It may be seen in successive generations, but it usually skips one or

more generations. The disease frequently affects more than one person in a family and involves males more frequently than females. The literature records 1 case in which microscopic examination of the optic nerve was made post mortem, by Rehsteiner in 1932. This author observed atrophy of the ganglion cells and the nerve fiber layer of the retina and atrophy of the optic nerve, limited to the papillomacular bundle, other parts of the nerve being normal. The medullary sheaths were almost all destroyed. There were an increase in the glia and atrophy of the finer connective tissue partitions of the nerve. Alexander observed 4 cases of Leber's disease in a family of 5 children. The cases were equally distributed between the sexes. In the 2 girls the onset of the disease was at 5 years of age, whereas usually the first symptoms occur between puberty and the age of 30 years. In these patients the condition did not improve. One patient presented an associated nystagmus; this is unusual in the presence of greatly reduced vision. No other record of its concurrence with Leber's disease could be found. The chief symptom is great diminution in vision with central scotoma. Complete blindness does not occur. The prognosis is poor, but improvement sometimes occurs. Treatment is of no benefit.

J. A. M. A.

ATMOSPHERIC AND IMMERSION BLAST INJURIES. FRANK V. THEIS, War Med. **4**:262 (Sept.) 1943.

Atmospheric and immersion high explosive blasts produce injuries to the internal structures of the exposed body with absence of external evidence of trauma. There may be symptoms referable to all parts of the body. With respect to the nervous system, headaches are common, and sensitivity to noise and tinnitus are fairly frequent. Changes in the personality are important and become most prominent several days after injury.

Relatively few cases of blast injury have been studied adequately at autopsy. The air-containing viscera seem primarily to be injured by blast. Injuries to the kidneys, spleen and liver have not been observed at autopsy, but animal experiments have demonstrated that all organs may be affected.

There is no essential difference between the injuries resulting from atmospheric blasts and those due to immersion blasts. The detonation of high explosives produces an extremely intense longitudinal compression wave, which moves forward, although the particles of the medium through which it passes do not move, but oscillate backward and forward. The wave has two phases—a pressure and a suction component. It travels four times as far in water as in air. The damaging effect of the blast wave is due largely to the sudden pressure of the external surface of the body on the internal structures.

In order to prevent the serious effects of blast on the body, the following protective measures may be taken:

1. In the presence of an impending blast, one should exhale forcibly and contract the abdominal muscles.

2. For atmospheric blasts, protection is afforded by shelters, heavy clothing and cotton or plastic appliances for the ears. It is important to lie face down in a trench or gutter.

3. In the presence of immersion blasts, one should keep the head, and as much of the body as possible, out of the water, by swimming on the back, and should keep the mouth open. It is advisable to wear a heavy kapok life jacket and delay as long as possible entering the water when immersion blasts are expected.

The treatment for blast injuries is symptomatic, and its appropriateness depends on the extent, severity and distribution of the injuries.

PEARSON, Philadelphia.

NIGHT BLINDNESS OF WAR. PAUL H. WOSIKA, War Med. **4**:331 (Sept.) 1943.

Malnutrition (lack of vitamin A) causes night blindness, xerosis and xerophthalmia. Therapeutic correction is simple, swift and sure. Although in the literature poor dark adaptation, as measured by instruments, has been included under the term night blindness, it is not related to it, and treatment with huge doses of vitamin A does not correct it.

PEARSON, Philadelphia.

APPLICATION OF ELECTROENCEPHALOGRAPHY IN THE NAVY IN WARTIME. ROBERT S. SCHWAB, War Med. **4**:404 (Oct.) 1943.

Ninety per cent of the naval personnel with clinical epilepsy have had abnormal electroencaphalograms. Electroencephalography has proved thoroughly useful in the diagnosis of, or in the elimination of a suspicion of, an epileptic tendency. The electroencephalographic examination, however, is not a short cut, nor does it eliminate the need for a careful history and neurologic study.

PEARSON, Philadelphia.

HYSTERICAL HOMONYMOUS HEMANOPSIA WITH HEMIPLEGIA AND HEMIANESTHESIA. SLOAN G. STEWART, GREG C. RANDALL and F. REGIS RIESENMAN, War Med. **4**:606 (Dec.) 1943.

The authors report the case of a man with homonymous hemanopsia associated with hemiplegia. Several examinations of the visual fields showed a constant field defect, consisting of homonymous macula-sparing hemanopsia consistent with a diagnosis of an organic lesion of the brain affecting the right optic pathway or tract. The subjective sensory and motor complaints were compatible with such involvement. The clinical course, the objective neurologic observations and the efficacy of psychotherapy indicated the hysterical condition. A review of the literature indicates that perhaps this is the first case of homonymous hemanopsia of a true hysterical nature ever to be reported.

PEARSON, Philadelphia.

ELECTROENCEPHALOGRAPHY IN THE ARMY GENERAL HOSPITAL. DANIEL SILVERMAN, War Med. **5**:163 (March) 1944.

On the basis of the study of the clinical records and electroencephalographic tracings for 173 neuropsychiatric patients, Silverman concludes that the electroencephalogram is of special value in the diagnosis and disposition of epilepsy, psychoneuroses resembling organic disease of the brain, syndrome following head injury and focal lesions of the brain from any cause.

PEARSON, Philadelphia.

TWO CASES OF RUBELLA MENINGO-ENCEPHALITIS. R. I. C. BRADFORD, Brit. M. J. **1**:312 (March 13) 1943.

The rarity of meningoencephalitis in association with rubella is evidenced by the fact that Taylor, in 1937, could find only 12 reported cases. Bradford reports a case of such an occurrence in a youth aged 19 years and another in a boy of 18 years. Both patients recov-

toms. There was no evidence of the presence of angioma of the retina on examination.

Case 4.—H. S. B., aged 19, a veteran of World War II, was admitted for treatment of a cerebellar tumor. He had been in good health until January 1941, when he began to experience continuous occipital headaches, which were intensified by his rising from a horizontal position, by lifting weights, by bowel movements and by jarring of the body. Nausea and episodes of nonprojectile vomiting of liquids and solids were manifested later. Visual disturbances, affecting the right eye more than the left, were common. At no time did the patient suffer from spells of unconsciousness or weakness of the extremities. He was admitted to the veterans hospital on May 5, 1944. Neurologic examination revealed the presence of bilateral papilledema, definite coarse nystagmus, with the quick component to the left, and ataxia involving his extremities. The deep reflexes of the upper and lower extremities were diminished. No pathologic reflexes were present. Examination of the spinal fluid and roentgenograms of the skull gave no evidence of any abnormality. The diagnosis was cerebellar tumor, probably hemangioblastoma because of the family history. A bilateral suboccipital craniotomy was performed on June 13, with intratracheal anesthesia. The dura over the cerebellar hemispheres was noted to be under greatly increased intracranial pressure, and removal of the tumor was impossible. The patient died six hours after operation. Autopsy, for verification of the type of tumor and the presence of angiomatous cysts in the retina or other parts of the body, was not permitted.

SUMMARY AND CONCLUSIONS

Tumors of the brain occurred in 4 members of one family—the father, aged 45; a daughter, aged 24, and 2 sons, aged 23 and 19. In 2 members of the family the lesion was a cerebellar hemangioblastoma; in the other 2 members the type of the neoplasm was not determined, but a cerebellar tumor, which could not be removed, was present in 1 of them. Examination of the retina did not reveal the presence of a coexisting angioma. Three of the 4 members of the family died of the cerebral tumor; the other member, a son aged 23, is living and well, after surgical intervention. Since no autopsy was performed, the presence of angiomatous cysts in other parts of the body could not definitely be ruled out. It is probable that the members of this family had Lindau's disease. One sister in the family is well and has not been afflicted with this condition. She is fully aware of the occurrence of tumor of the brain in her family and has sought neurologic examination from time to time as a precautionary measure.

3145 North Cambridge Avenue, Chicago.

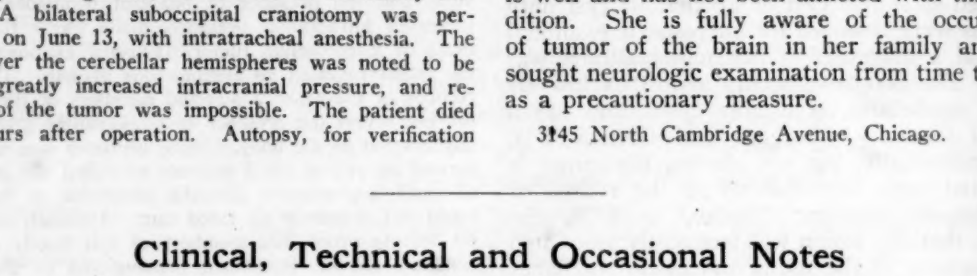
Clinical, Technical and Occasional Notes

HANDY AND INEXPENSIVE TEMPERATURE TESTER

Thomas Edwin Bamford Jr., M.D., New York

Examination of heat and cold sensibility is often neglected during a routine neurologic examination because the tubes used for this test are bulky and unhandy. The tubes on the market for this purpose are also expensive.

By accident, it was discovered that the tube illustrated here,[1] which has been placed on the market as a sterile container for hypodermic syringe and needle, will serve the purpose well. It is inexpensive (costing 50 cents) and handy, and can be chilled or heated to the desired temperature. It is 15.5 cm. long and 4.5 cm. in circumference at the open end and tapers to a point. As it is plated and smooth, it does not radiate. This tube can be carried in a small container, and the rubber cap affords a firm grip at any temperature.



Tube, with rubber cap.

1. Steritube, manufactured by Becton, Dickinson & Company, Rutherford, N. J.

572 Park Avenue.

Abstracts from Current Literature

EDITED BY DR. BERNARD J. ALPERS

Psychiatry and Psychopathology

LATE EFFECTS OF LEAD POISONING ON MENTAL DEVELOPMENT. R. K. BYERS and E. E. LORD, Am. J. Dis. Child. **66**:471 (Nov.) 1943.

According to Byers and Lord, it has not been generally recognized that lead poisoning occurring in early life usually has a disastrous effect on mental development. They present a follow-up study of 20 school children who had been hospitalized in infancy or early childhood because of lead poisoning. None of them exhibited striking evidence of encephalopathy during their primary admission, and all were adjudged to have made a complete recovery from lead poisoning when discharged from the original hospitalization. The authors emphasize the length of the cycle of ingestion, the storage and elimination of lead in relation to the relatively short duration of the symptoms and the signs allowing a diagnosis of lead poisoning. It is probable that lead poisoning of the sort discussed here can at present be recognized in only a small percentage of cases. Failure of the normal processes of growth and development of the cortex prevented all but 1 of the 20 children from progressing satisfactorily at school. In addition to difficulties in the general intellectual and sensorimotor spheres, other evidence of interference with the normal development of the nervous system was present. Recurrent convulsions appeared in 3 of the children, at 4, 4½ and 5½ years of age respectively. One girl who had peripheral neuritis as a baby acquired a positive Babinski sign, and a boy who was discharged from the hospital as well at about 3 years of age had hyperactive reflexes and sustained clonus of the left ankle when reexamined at 9 years of age. Behavior difficulties were common throughout the series. Much of this behavior could be classified as "forced reaction to stimuli in the environment," described by Strauss and Werner as an evidence of cortical damage. It was apparently the result of loss of the normal inhibitory function, thought to reside in the cortex. It was usually described as unreliable impulsive behavior, cruel impulsive behavior, short attention span and the like.

J. A. M. A.

ALTERATIONS IN COMMUNICABILITY, CONTENT OF THOUGHT, AND AFFECTIVE RESPONSE DURING IRRITATIVE (CAMPHOR) THERAPY. EMERICK FRIEDMAN, J. Nerv. & Ment. Dis. **98**:229 (Sept.) 1943.

Friedman studied the alterations in the productivity of 70 previously noncommunicative psychotic patients who were given camphor therapy. In the preparatory phase, the selection of a group of patients, their segregation from others and the excitement of the treatment fostered an *esprit de corps* among them which resulted in a banding together, but did not change the nature of their thought content. The injection of camphor induced irritative delirium with a feeling of impending annihilation. In practically every patient, even those with a previously acute psychosis, there was produced a verbal response communicating the necessity for haste and quick preventive action. At first symbolic implements were used, but with succeeding treatments an increasingly direct and understandable speech was employed. Many of the patients began to write letters for the first time since institutionalization. Disturbances in equilibration were noted frequently, in the nature of experiences of rapid alterations of equilibrium. These were accompanied by terrifying feelings of disorganization. Somatic complaints usually centered about the thorax and abdomen, while actual skeletal injuries, such as sprains, or even fractures, were disregarded. Various motor manifestations of sexual activity were observed during the delirium. These included masturbatory and heterosexual and homosexual pantomimes. Several patients revealed vague sadistic or masochistic trends. Pathologic sexuality was also released verbally. Almost every patient expressed experiences of body alterations that might be classified as castration fears. Even in previously apathetic patients, an affective appropriateness was called forth, as well as increased sensitivity and sudden impulsive destructive and self-destructive outbursts.

The author suggests that these alterations in productivity and affect could be employed in psychotherapeutic efforts accompanying the irritative therapy.

CHODOFF, Langley Field, Va.

MENTAL SYMPTOMS IN MULTIPLE SCLEROSIS. CARL SUGAR and RAYMOND NADELL, J. Nerv. & Ment. Dis. **98**:267 (Sept.) 1943.

Sugar and Nadell review the literature on the mental symptoms of multiple sclerosis, emphasizing the great diversity of observations by different authors. They attribute this primarily to the difference in the source of the material studied, this being exclusively either from neurologic hospitals or from institutions for mental disease.

They report a study of 28 patients with multiple sclerosis admitted to a hospital for chronic neurologic conditions. They found euphoria to be the prevailing mood in 15 patients, dysphoria in 5 and fluctuating moods in 8 patients. As a change from their prevailing mood prior to the onset of the illness, 11 patients showed an increase in cheerfulness; 10 of these were basically cheerful. Seven patients showed an increase in depression; in 4 this was the prevailing mood. Six patients showed an increased variability of mood; of these, all but 1 had always had variable moods. Four patients displayed no change. Thus, in the majority of patients the prevailing mood was accentuated. In all but 2 of the patients the affective coloring of the thought content was similar to that of the prevailing mood. A sense of physical well-being accompanied the mood in the euphoric patients, whereas the dysphoric patients felt bad most of the time. Exaggerated emotional expression was present in 79 per cent, while 21 per cent of the patients showed little outward expression of emotion. Loss of or imperfect control of affective expression was found in 18 patients (64 per cent); 16 of these stated that their outward expression was incongruous with their emotional feeling.

The authors corroborate the contention of Cottrell and Wilson that change in the prevailing mood, in emotional

expression and control and in the sense of physical well-being constitutes a diagnostic triad which occurs more frequently than any single neurologic sign. They believe that the affective disturbances in mood and behavior fit in with the patient's previous personality makeup.

CHODOFF, Langley Field, Va.

WORK AND THE PLEASURE PRINCIPLE. IVES HENDRICK, Psychoanalyt. Quart. **12**:311, 1943.

Hendrick formulates the theory that work pleasure is not primarily displaced or sublimated sensual pleasure, but that it is the pleasure afforded by effective integration of the neuromuscular and intellectual functions. He calls the need of human beings for such pleasure the work principle, and he regards this principle as an expression of the instinct to master, the goal of which is control or alteration of environmental situations through the effective development of integrated intellectual and motor functions. The first objective evidence of the work principle is to be observed in the development of each partial function in infancy, when the stereotyped repetition of the function is replaced by the ability to modify the function effectively and to adapt it to tasks which satisfy the need to master. The work principle should therefore be regarded as evidence of maturity of ego function, and the repetition compulsion, as evidence that the ego is functioning inadequately for the skilful performance of a certain task.

PEARSON, Philadelphia.

CONCERNING THE PSYCHOGENESIS OF CONVULSIVE DISORDERS. LEO H. BARTEMEIER, Psychoanalyt. Quart. **12**:330, 1943.

Bartemeier believes that convulsions constitute a means of discharging destructive energy autoplastically. While all human beings have this innate preformed possibility of discharge, the predisposition of those who finally manifest a convulsive tendency consists in their predilection for the convulsive way of discharge. If the energy quantum is high enough and other ways are blocked, even the nonpredisposed person will turn to convulsions in order to relieve the mental apparatus of tension, and to a minor degree every person will show convulsive manifestations in one way or another.

PEARSON, Philadelphia.

FEAR OF DEATH. GREGORY ZILBOORG, Psychoanalyt. Quart. **12**:465, 1943.

Morale has to do with the avoidance of turning aggression against one's self and with the proper direction of aggression outward. The fundamental psychologic issue involved in the problem of morale is that of how one reacts to the fear of death, which is present constantly in mental functioning. It must be kept repressed, and in order to increase the repression one spends much time and energy in denying the possibility of death and in trying to master it. One's interest in news of violent deaths overlays an unconscious egocentricity. "It is not I who was killed."

When war comes to a civilized community, the fear of death, so well taken care of in peacetime, undergoes a crucial change. The civilian may still utilize the mechanism which makes him feel he is an exception and therefore cannot be killed. Not until members of his family and friends fall in battle does he come to grips with the fear of death by way of identification with those who were killed. Therefore compassion, an expression of the unconscious sense of guilt about those who have fallen, and scorn, a momentary sense of anger and hatred for the enemy who kills, become prominent components of the complex set of psychologic factors that group themselves around the repressed fear of death. Compassion and scorn become the core of morale, and the actual fear of death is mastered through hatred of the enemy. In the civilian who is exposed to bombing the fear of death is activated, but is countered by an increase in religious feeling and the mobilization of hatred. In this way the fear of death is transformed into a sadomasochistic combination, which on the socialized level is represented by awareness of grim hatred and of readiness to make great sacrifices to win the war. The accentuation of murderous drives is helpful in war, but if it occurred in times of peace it would lead to depressive states and to an increase in the rate of suicide. The rate of suicide is always higher in peacetime.

On the battlefield green troops become seasoned soldiers as they become angry, that is, as they convert their fear of death and hatred into hatred and aggression. In that fear of death which would be called neurotic, i. e., in the war neuroses, the victim hates, but he has a paralysis of motor aggression. He is unable to fight because he identifies himself with the dead as a result of a severe sense of guilt which antedates his military service. Those who are the most conspicuous misfits on the battlefront are the most ethically sensitive people, although they are socially the most useless.

Civilian morale might be improved if the general population were better informed about the losses and could see more of the wounded.

PEARSON, Philadelphia.

BODY AS PHALLUS: A CLINICO-ETYMOLOGICAL NOTE. HENRY ALDEN BUNKER, Psychoanalyt. Quart. **12**: 476, 1943.

The English word "body" is of unknown origin. Middle Gaelic, however, has the word bod, meaning penis. This corresponds to the psychoanalytic observation that body and penis are interchangeable in the unconscious. Bunker cites the fantasy of a 6 year old boy, which illustrates clearly that for this boy the word "body" meant penis. PEARSON, Philadelphia.

THE PSYCHOANALYTIC CONCEPT OF MEMORY AND ITS RELATION TO RECENT MEMORY THEORIES. ERNST LEWY and DAVID RAPAPORT, Psychoanalyt. Quart. **13**:16, 1944.

The psychoanalytic theory of memory is based on the view that memory traces are used by psychic forces, which find expression through them. The modes in which strivings make use of memory material vary significantly among such phenomena as everyday remembering, dreams and remembering of childhood experiences. The theories of memory deduced from experimental work by Lewin, Koffka and Bartlett show a striking parallel with the psychoanalytic theory.

PEARSON, Philadelphia.

HYPOGLYCEMIA AND TENSION-DEPRESSION. T. A. C. RENNIE and J. E. HOWARD, Psychosom. Med. **4**: 273 (July) 1942.

Rennie and Howard report on a series of 7 patients who had symptoms of a psychiatric disorder characterized chiefly by tension and colored by manifestations of depression. In addition, all the patients exhibited, as a conspicuous feature, a craving for food, which when satisfied resulted in temporary alleviation of the

emotional disturbances. Six of the patients were women, and 1 was a man. The hypoglycemic symptoms were in keeping with the flat dextrose tolerance curves obtained for all the patients. The hypoglycemia appeared to be secondary to the psychiatric disorder, since the former disappeared after the treatment of the latter. The authors emphasize that, since there are wide variations in the level of the blood sugar outside the accepted normal range, the dextrose tolerance test should be properly interpreted in terms of the individual personality. They conclude that when the specific complaints suggestive of hypoglycemia are presented, it is important to study the total personality and, in a certain group of patients, to treat the personality disturbances primarily.

SCHLEZINGER, Philadelphia.

PHYSIOLOGIC AND PSYCHOLOGIC STUDIES IN SPONTANEOUS HYPOGLYCEMIA. J. ROMANO and G. P. COON, Psychosom. Med. **4**:283 (July) 1942.

Romano and Coon describe the case of a married man aged 44 in which the original diagnosis of hysterical fugue was later proved incorrect and was replaced by a pathologically verified diagnosis of hypoglycemia due to a benign islet cell adenoma of the pancreas. The patient was an emotional, rigid, sensitive and dependent person who evidenced considerable repressed aggression. His illness was also characterized by recurrent episodes of confusion, uninhibited emotional behavior and disconnected movements. After the surgical removal of the adenoma the episodes were eliminated, but the essential nature of his personality remained unchanged.

The authors believe that the principal source of error in the diagnosis of a neurotic personality lies in the methods directed toward exclusion of positive data related to physical disease or psychosis. The need for the establishment of positive criteria of neurotic personality structure is emphasized.

In considering the relation between increased cerebral metabolism as a result of hypoglycemia and the resultant disturbances in intellectual, emotional and motor behavior, the authors suggest that inhibition and repression may be the physiologic and psychologic expressions of a basic phenomenon. The release of concrete intellectual activity, of less inhibited emotional expression and of less integrated neurologic behavior may result in all delirious patients as it did in this hypoglycemic patient. As in all deliriums, the primary psychologic disturbance is situated at the level of awareness. This disturbance of consciousness is more or less impersonal. On the other hand, the emotional behavior, which is dependent on the degree of disturbance of consciousness, is more individualized and specific in reflecting the personality structure of the delirious patient.

SCHLEZINGER, Philadelphia.

A PERSONALITY STUDY OF ALCOHOL ADDICTION. CHARLES C. HEWITT, Quart. J. Stud. on Alcohol **4**:368 (Dec.) 1943.

Hewitt employed the Minnesota multiphasic personality schedule and the Pressey senior classification test in an effort to determine the personality of persons addicted to alcohol. He claims that the schedule employed appears to be superior to any other inventory.

The case material consisted of 37 members of Alcoholics Anonymous, Inc., 15 other persons with alcoholism and 12 subjects who enjoyed the effects of alcohol but rarely drank in excess. The author concludes that addiction to alcohol seems to accompany, with few exceptions, deep personality disorders. Nearly all of the alcohol addicts, according to this study, exhibited a "marked psychopatic deviation which was often associated with neurotic, paranoid or schizoid trends."

GUTTMAN, New York.

PHYSICAL TREATMENT OF ACUTE PSYCHIATRIC STATES IN WAR. WILLIAM SARGANT, War Med. **4**:577 (Dec.) 1943.

Sargant points out that persons with reasonably good previous personalities do not generally break down or give up the fight against neurotic symptoms if their physical health is good. In treatment of acute conditions he emphasizes the need for sedation, preferably administration of the barbiturates intravenously or of paraldehyde by mouth. Forced feeding of enormous quantities of food and liquids is necessary with patients who have lost weight. This may be supplemented by small doses of insulin or by the Weir Mitchell technic. Sargant ends with the statement "Breakdown, even in the presence of great physical exhaustion and deterioration, is less common in an advancing triumphant army."

PEARSON, Philadelphia.

MANUAL OF EMERGENCY TREATMENT FOR ACUTE WAR NEUROSES. LAWRENCE S. KUBIE, War Med. **4**: 582 (Dec.) 1943.

Most war neuroses begin as acute disorders. These disturbances frequently develop gradually; i. e., the collapse occurs only after cumulative stress. Paramount in this stress are the effects of fatigue and hunger, of incessant and increasing danger and of a series of repeated, rapidly successive, narrow escapes. It takes time to recover from the effects of sudden danger; but when one threat is followed at once by another, there is no interval in which the emotional reverberations can die out, and the person spends his days and nights living as though he were engaged in an incessant struggle to escape from or master the original danger. Each successive escape contributes to the secretly mounting tension. At some point the tension becomes manifest, usually the first sign being a specific disturbance of sleep and terror dreams. Once sleep is disturbed, fatigue increases rapidly, giving rise to moments in the waking hours in which the patient lapses into momentary half-dissociated states. At such moments sudden movements, noises or lights cause a startled panic. As the fatigue and moments of terror spread through the day, the patient becomes unstable and depressed and feels alienated from his comrades. There are also many subtle changes in his daily behavior.

As any man may break under sufficient stress, prevention depends on the recognition of incipient stages and on steps which can be taken to stave them off. There are several important preventive measures: 1. Officers and men should be taught to recognize early any disturbances of sleep. 2. As soon as sleep disturbances are noted, sedation, with a combination of two sedatives, should be given; one of the sedatives should act rapidly, and the other should act slowly and have a sustained action. 3. A low light should be kept burning in the sleeping quarters throughout the night. 4. The soldier should be awakened swiftly and be given a cold drink or black coffee, followed by amphetamine sulfate, as soon as he is awake. 5. Rest periods during the day should not be silent, but the soldier should be kept amused and interested.

After the neurosis is established, the emergency treatment is as follows: (1) Controlled sleep, physical restoration and friendly group companionship; (2) superficial catharsis through companionship in isolation, "gentling" and opportunity for cathartic release; (3) prolonged sleep under continuous narcosis; (4) group methods of psychotherapy; (5) deconditioning procedures; (6) hyponarcoanalysis and the induced hypnagogic reverie, and (7) in cases in which the patient is inaccessible to any contact, even under narcosis, one or two electric shock treatments or sustained periods of hypoglycemia, without coma or convulsions.

PEARSON, Philadelphia.

PSYCHOSES IN OFFICERS IN WORLD WAR II. ADDISON M. DUVAL, War Med. **5**:1 (Jan.) 1944.

Duval reports on the study of 100 officers admitted consecutively to St. Elizabeths Hospital and compares this group with a somewhat similar group of officer patients in World War I.

Twenty-seven patients gave a history of previous mental illness, and 10 had previously been treated in hospitals for mental disease. At least 20 of the 27 patients would have been rejected on induction if they had been given an adequate psychiatric examination. The ages of the entire group varied from 20 to 49 years. Eleven men had service of less than one month, and 45, of less than one year. In many instances situational factors were important in producing the psychotic attack. Thirty-four patients had manic-depressive psychoses, and 30, schizophrenia. With 10 patients electric shock gave good results. Seventy-four patients have been discharged from the hospital, 49 as recovered or socially recovered, 20 as showing improvement, 2 as showing no improvement and 3 as free from psychosis.

PEARSON, Philadelphia.

HYSTERICAL VISUAL DEFECTS. PAUL T. MCALPINE, War Med. **5**:129 (March) 1944.

McAlpine reports briefly 9 cases of amblyopia of hysterical origin and 1 case of amblyopia regarded as a symptom of dementia precox. Hysterical amblyopia is of infrequent occurrence and must be differentiated from retrobulbar neuritis, toxic neuritis and amblyopia due to disease of the central nervous system.

PEARSON, Philadelphia.

RETROGRADE AMNESIA. W. MAYER-GROSS, Lancet **2**: 603 (Nov. 13) 1943.

Mayer-Gross tested retrograde amnesia in 102 trials on 46 patients. The subjects were all psychotic, without impairment of memory; the author excluded patients whose full cooperation was doubtful. Tests were made for memory of a series of four picture cards the minute prior to the administration of electric shock therapy. After twenty-four hours or less the patient was tested for memory recall and for recognition. Only 2 patients recalled all cards, and of all 102 tests only 29 resulted in complete recognition. The author stated that this result indicates that retrograde amnesia is not a universal symptom after epileptiform convulsions. There was a direct relation between the time the pictures were shown and recall and recognition; the pictures shown first were better remembered than those shown next, and the pictures shown last were least often recalled or recognized. Thus the defect was true retrograde amnesia. The results of testing three to eight hours after the shock were compared with those of tests made twenty-four hours later. The first picture shown was consistently better remembered a few hours after the seizure than a full day later. The author concluded that this observation indicated that "it is not a matter of indifference at which time the patient is tested for retrograde amnesia." In only one tenth of the experiments did the retrograde amnesia last longer than one minute.

Mayer-Gross discusses the theories explaining this phenomenon. The most popular current theory among neurologists and psychiatrists is that the traces, or engrams, of what has gone before the accident are destroyed. The author believes that the difference between recall and recognition in his tests is too great to support this theory by which he means that one could not recognize so well what one could not recall if the subject matter had been actually destroyed or blotted out. Rather, he prefers the theory that the consolidation of recent memories is interrupted; there is a retroactive inhibition similar to that known to normal psychology.

He cannot explain the individual differences in retrograde amnesia found among his subjects. In patients with head injuries the variations might be explained by differences in local cerebral damage, but the duration of electric shock and its clinical symptoms vary so little from subject to subject "that one is forced to surmise the presence of an unknown personal factor." Nor can he explain the clinical fact that patients in post-traumatic drowsiness will describe an accident in detail which they cannot remember when completely conscious later. This phenomenon seems to parallel the fuller recollection of the first picture in the early test of his series.

MCCARTER, Philadelphia.

Diseases of the Brain

INCIDENCE OF THE CHANGES IN THE RETINAL VEINS IN MULTIPLE SCLEROSIS. J. V. TRUESCH and C. W. RUCKER, Proc. Staff Meet., Mayo Clin. **19**:253 (May 17) 1944.

Rucker describes perivenous sheating in the retinal veins in cases of multiple sclerosis and asserted that in most of the cases in which this sign was present a definite or a presumptive diagnosis of multiple sclerosis had been made.

Truesch and Rucker report on the examinations of the fundus in 52 patients with multiple sclerosis, in all of whom careful search was made for perivenous sheathing. All the examinations were made through dilated pupils; all observations were confirmed, and all borderline cases were discarded. Of the 52 patients studied, 10 (19 per cent) showed the changes described as perivenous sheathing. The ages of the 10 patients varied from 18 to 51, 5 being women and 5 men. The average time of appearance of the sheathing after the first symptom of multiple sclerosis was four and one-third years. The condition is not related to the occurrence of pallor of the optic disks nor to a history of previous blurring of vision.

ALPERS, Philadelphia.

LEBER'S DISEASE: REPORT OF FOUR CASES IN ONE FAMILY. C. S. ALEXANDER, Texas State J. Med. **39**:301 (Sept.) 1943.

Leber's disease is defined as hereditary bilateral primary optic nerve atrophy. It is transmitted almost entirely by the female, who is usually unaffected, although it may rarely be transmitted by the male directly or indirectly through the daughters. It may be seen in successive generations, but it usually skips one or

more generations. The disease frequently affects more than one person in a family and involves males more frequently than females. The literature records 1 case in which microscopic examination of the optic nerve was made post mortem, by Rehsteiner in 1932. This author observed atrophy of the ganglion cells and the nerve fiber layer of the retina and atrophy of the optic nerve, limited to the papillomacular bundle, other parts of the nerve being normal. The medullary sheaths were almost all destroyed. There were an increase in the glia and atrophy of the finer connective tissue partitions of the nerve. Alexander observed 4 cases of Leber's disease in a family of 5 children. The cases were equally distributed between the sexes. In the 2 girls the onset of the disease was at 5 years of age, whereas usually the first symptoms occur between puberty and the age of 30 years. In these patients the condition did not improve. One patient presented an associated nystagmus; this is unusual in the presence of greatly reduced vision. No other record of its concurrence with Leber's disease could be found. The chief symptom is great diminution in vision with central scotoma. Complete blindness does not occur. The prognosis is poor, but improvement sometimes occurs. Treatment is of no benefit.

J. A. M. A.

ATMOSPHERIC AND IMMERSION BLAST INJURIES. FRANK V. THEIS, War Med. **4**:262 (Sept.) 1943.

Atmospheric and immersion high explosive blasts produce injuries to the internal structures of the exposed body with absence of external evidence of trauma. There may be symptoms referable to all parts of the body. With respect to the nervous system, headaches are common, and sensitivity to noise and tinnitus are fairly frequent. Changes in the personality are important and become most prominent several days after injury.

Relatively few cases of blast injury have been studied adequately at autopsy. The air-containing viscera seem primarily to be injured by blast. Injuries to the kidneys, spleen and liver have not been observed at autopsy, but animal experiments have demonstrated that all organs may be affected.

There is no essential difference between the injuries resulting from atmospheric blasts and those due to immersion blasts. The detonation of high explosives produces an extremely intense longitudinal compression wave, which moves forward, although the particles of the medium through which it passes do not move, but oscillate backward and forward. The wave has two phases—a pressure and a suction component. It travels four times as far in water as in air. The damaging effect of the blast wave is due largely to the sudden pressure of the external surface of the body on the internal structures.

In order to prevent the serious effects of blast on the body, the following protective measures may be taken:

1. In the presence of an impending blast, one should exhale forcibly and contract the abdominal muscles.

2. For atmospheric blasts, protection is afforded by shelters, heavy clothing and cotton or plastic appliances for the ears. It is important to lie face down in a trench or gutter.

3. In the presence of immersion blasts, one should keep the head, and as much of the body as possible, out of the water, by swimming on the back, and should keep the mouth open. It is advisable to wear a heavy kapok life jacket and delay as long as possible entering the water when immersion blasts are expected.

The treatment for blast injuries is symptomatic, and its appropriateness depends on the extent, severity and distribution of the injuries.

PEARSON, Philadelphia.

NIGHT BLINDNESS OF WAR. PAUL H. WOSIKA, War Med. **4**:331 (Sept.) 1943.

Malnutrition (lack of vitamin A) causes night blindness, xerosis and xerophthalmia. Therapeutic correction is simple, swift and sure. Although in the literature poor dark adaptation, as measured by instruments, has been included under the term night blindness, it is not related to it, and treatment with huge doses of vitamin A does not correct it.

PEARSON, Philadelphia.

APPLICATION OF ELECTROENCEPHALOGRAPHY IN THE NAVY IN WARTIME. ROBERT S. SCHWAB, War Med. **4**:404 (Oct.) 1943.

Ninety per cent of the naval personnel with clinical epilepsy have had abnormal electroencaphalograms. Electroencephalography has proved thoroughly useful in the diagnosis of, or in the elimination of a suspicion of, an epileptic tendency. The electroencephalographic examination, however, is not a short cut, nor does it eliminate the need for a careful history and neurologic study.

PEARSON, Philadelphia.

HYSTERICAL HOMONYMOUS HEMANOPSIA WITH HEMIPLEGIA AND HEMIANESTHESIA. SLOAN G. STEWART, GREG C. RANDALL and F. REGIS RIESENMAN, War Med. **4**:606 (Dec.) 1943.

The authors report the case of a man with homonymous hemanopsia associated with hemiplegia. Several examinations of the visual fields showed a constant field defect, consisting of homonymous macula-sparing hemanopsia consistent with a diagnosis of an organic lesion of the brain affecting the right optic pathway or tract. The subjective sensory and motor complaints were compatible with such involvement. The clinical course, the objective neurologic observations and the efficacy of psychotherapy indicated the hysterical condition. A review of the literature indicates that perhaps this is the first case of homonymous hemanopsia of a true hysterical nature ever to be reported.

PEARSON, Philadelphia.

ELECTROENCEPHALOGRAPHY IN THE ARMY GENERAL HOSPITAL. DANIEL SILVERMAN, War Med. **5**:163 (March) 1944.

On the basis of the study of the clinical records and electroencephalographic tracings for 173 neuropsychiatric patients, Silverman concludes that the electroencephalogram is of special value in the diagnosis and disposition of epilepsy, psychoneuroses resembling organic disease of the brain, syndrome following head injury and focal lesions of the brain from any cause.

PEARSON, Philadelphia.

TWO CASES OF RUBELLA MENINGO-ENCEPHALITIS. R. I. C. BRADFORD, Brit. M. J. **1**:312 (March 13) 1943.

The rarity of meningoencephalitis in association with rubella is evidenced by the fact that Taylor, in 1937, could find only 12 reported cases. Bradford reports a case of such an occurrence in a youth aged 19 years and another in a boy of 18 years. Both patients recov-

ered. Encephalitis associated with exanthems has been attributed to the virus of the exanthem in question; to a virus common to all patients, normally latent, but activated by the exanthem, and to an allergic reaction in the brain following the general eruption. Bradford seems to favor the last theory.

ECHOLS, New Orleans.

HEAD INJURIES IN MOTOR CYCLISTS WITH SPECIAL REFERENCE TO CRASH HELMETS. H. CAIRNS and H. HOLBOURN, Brit. M. J. **1**:591 (May 15) 1943.

Cairns and Holbourn, in an extensive analysis of 106 cases of head injury sustained by motor cyclists wearing crash helmets, noted the following significant observations: The degree of injury varied from mild to severe. The injury of the scalp and skull corresponded with the site of the blow on the helmet. In 40 per cent of 81 cases the victim sustained multiple injuries to the head. Over half the blows occurred on the front of the helmet, and the crown received the least number of blows. The authors noted that in motor cyclists cerebral injury was almost invariably associated with a blow on the head, protection against which could be provided by a properly fitted crash helmet; only rarely was the brain damaged by a blow limited to the face. The wearing of the crash helmet reduced the incidence of fracture of the skull, decreased the degree of concussion and altered the severity of the injury in the direction of mildness. A comparison of the two types of crash helmets revealed the superiority of the pulp over the vulcanized rubber helmet.

ECHOLS, New Orleans.

ARSENICAL ENCEPHALOPATHY. R. B. NELSON, C. MCGIBBONS and F. GLYN-HUGHES, Brit. M. J. **1**: 661 (May 29) 1943.

Hemorrhagic encephalopathy is one of the rarest and most fatal complications occurring during the treatment of syphilis with arsenical compounds. The reason for its occurrence is not definitely understood. The dose and toxicity of the drug seem to bear no relation to its production. The authors consider as possible etiologic factors the nature of the syphilitic infection, vitamin deficiency and mental stress and strain. They believe that the drug has a direct toxic action on the cerebral capillaries. They regard pregnant women as "potential reactors." The clinical picture is one of cerebral irritation, which may be preceded by symptoms of headache and dizziness. Four cases are reported, in 3 of which the complication was fatal. In the other case recovery was attributed to repeated spinal drainage and the use of morphine.

ECHOLS, New Orleans.

NEUROHEPATIC DEGENERATION. T. FRACASSI, Rev. argent.-nort. de cien. méd. **1**:310 (July) 1943.

Fracassi shows that, contrary to Wilson's and von Economo's belief, the degenerative process in hepatolenticular degeneration is not limited to the lenticular nuclei and the putamen, but extends to the cerebral cortex, the white substance and the gray nuclei. He states therefore that neurohepatic degeneration would be a better term and includes under it not only Wilson's disease and the pseudosclerosis of Westphal and Strümpell but all cerebral degenerative alterations caused by degenerative hepatic lesions. A case is described in which the condition could be considered Wilson's disease but was really atrophic cirrhosis of Laënnec, probably due to a gastrointestinal disorder, since alcoholism or syphilis was absent. The patient showed neurologic manifestations ten years after onset of the first symptoms of hepatic cirrhosis. The first neurologic manifestation was mild paralysis of the hands and legs. Later, speech became slow and hesitant, and tremor of the hands developed. Clinical examination revealed an extrapyramidal syndrome, with parkinsonian rigidity of the face and tremor of the hands, moderate muscular rigidity and dysarthria and tremor of the lips and tongue with difficulty in swallowing. There were also signs of a pyramidal syndrome: pronounced decrease in the strength of limbs, exaggerated tendinous reflexes, bilateral clonus of the foot and a positive Babinski sign in the left foot. The patient also presented symptoms of cortical excitation in the form of epileptiform fits. He died of pneumonia, and necropsy showed atrophic cirrhosis of the liver with fibrous perihepatitis and interstitial splenitis, as well as varices of the esophagus. There were atrophy of the cerebral cortex, laminar softening and atrophy of the nuclei of the striatum, with softening and cystic dilatation of the perivascular spaces and areas of softening in the optic layer. Glial elements were abundant in the cortex and in the nuclei of the striatum. There were areas of circumscribed and diffuse loss of nuclei in the frontoparietal white substance.

J. A. M. A.

ACTION OF CARBON DIOXIDE IN EPILEPSY AND SCHIZOPHRENIA ON BASIS OF ELECTROENCEPHALOGRAPHIC OBSERVATIONS. A. F. KORNMÜLLER, München. med. Wchnschr. **89**:30 (Jan. 9) 1942.

Kornmüller investigated the influence of carbon dioxide on the electroencephalographic changes in patients with epilepsy and with dementia precox. The patients were subjected to electroencephalographic study while they breathed alternately ordinary air and 5 or 3 per cent carbon dioxide in pure oxygen. These studies were made on 40 patients, most of whom had either epilepsy or dementia precox. The observations demonstrated that carbon dioxide may decrease or completely counteract the abnormal electroencephalographic manifestations of these diseases. Carbon dioxide, especially its deficiency (hypocapnia), acts directly on the brain, inasmuch as it is an adequate stimulus for certain parts of the brain. Carbon dioxide increases the perfusion of blood in the brain, whereas its lack reduces the cerebral circulation. Carbon dioxide also plays a part in tissue respiration. Further investigations are necessary to determine which of the factors assumes the decisive role. The author's observations indicate that some patients with epilepsy or with dementia precox are more or less continuously, and not during hyperventilation alone, in an abnormal state, which can be counteracted by carbon dioxide. Electroencephalography indicates that this condition involves chiefly parts of the frontal area of the brain which are connected with the midbrain and the hypothalamus. It is possible that these parts of the brain are diseased or that carbon dioxide represents an adequate stimulus for some of them. This is also indicated by the effect of voluntary hyperventilation in patients with epilepsy. It is probable that therapeutic effects may be expected from the use of carbon dioxide in treatment of epilepsy, dementia precox and other diseases of the brain. The effects of metrazol, insulin and electric shock treatments may be partly due to the fact that these methods increase the carbon dioxide content of the brain and blood.

J. A. M. A.

Peripheral and Cranial Nerves

THE GUILLAIN-BARRÉ SYNDROME. R. FINLEY GAYLE and DALE GROOM, J. Nerv. & Ment. Dis. **98**:488 (Nov.) 1943.

Gayle and Groom review briefly the literature and the characteristic clinical picture of the Guillain-Barré syndrome. They report the case of a 4 year old child in whom complete flaccid quadriplegia, paralysis of the left abducens nerve, distended bladder, generalized hyperesthesia and absence of deep reflexes appeared three or four days after apparent recovery from an infection of the upper respiratory tract, in December 1942. A spinal tap on the fourth day in the hospital revealed 4 cells per cubic millimeter and a total protein content of 120 mg. per hundred cubic centimeters. Treatment according to the Kenny method was instituted, with local hot applications, manipulation, massage, exercises and use of removable casts. Recovery was progressive, and by September 1943 only some residual weakness and atrophy of the lower extremities remained. The authors believe the Kenny method to be of value in the treatment of this condition.

CHODOFF, Langley Field, Va.

CAUSES OF PAIN IN FEET AFTER PROLONGED IMMERSION IN COLD WATER. JAMES C. WHITE and SHIELDS WARREN, War Med. **5**:6 (Jan.) 1944.

Severe pain is common in the early and late stages of recovery from the results of prolonged immersion of the feet in cold water. On the basis of microscopic and other studies, White and Warren conclude that in the early phase of inflammation the pain is due to anoxia of the injured superficial tissues and nerve endings. Pain of this sort can be controlled by cooling the legs, as this procedure lowers cellular metabolism and makes the reduced demand for oxygen commensurate with the limited supply which can be furnished by the thrombosed superficial blood vessels.

After the period of inflammation has passed, aching pain and rigidity of the toes may cause prolonged incapacity in persons who have had more severe injury. This pain and rigidity are due to an increase in interstitial connective tissue and collagen. The nerves are embedded in fibrous tissue and show endoneural fibrosis. Pain of this type tends to ease after six to eight months, the time at which the collagen surrounding the nerves ceases to contract.

PEARSON, Philadelphia.

Society Transactions

CHICAGO NEUROLOGICAL SOCIETY

R. P. MACKAY, M.D., *President, in the Chair*

Regular Meeting, Feb. 8, 1944

Intracranial Chordoma: Report of a Case. DR. PETER BASSOE.

A single woman aged 32, a private secretary, was seen by me in 1937 because of mild paralysis agitans involving the right side which had come on six years before without any signs of encephalitis or other acute infection. I did not see her again until January 1942, when the paralysis agitans was no worse but she complained of sore eyes. The vision and eyegrounds were normal. In February 1943 she complained for the first time of getting tired and of more difficulty with the right hand. On April 1, 1943 she awoke with severe headache on the left side and triplopia on looking to the left. When she was seen on April 5, the presence of triplopia was verified, and the left rectus muscle was about 30 per cent paretic. The headache was relieved by codeine. On admission to the hospital on April 6, she still had the triplopia but little headache. Examination by Dr. E. V. L. Brown on the day after admission revealed only diplopia. The palsy of the external rectus muscle remained, with the width between the images constantly changing. The pupils and the disks were normal. In a few days the palsy of the external rectus muscle almost disappeared, and the patient saw double only on looking to the far left. She left the hospital on April 10. Aside from the diplopia, the neurologic condition was unchanged. She was in her usual condition, free from diplopia, until the last week in July, when slight palsy of the left external rectus muscle reappeared, and she also complained of headache in the left frontal region. The weakness of the external rectus muscle slowly increased, to be complete by the end of August. In late September the tongue and the inside of the mouth on the left side became hypalgesic, and in the course of a month the whole area of distribution of the left trigeminal nerve was analgesic. There was much headache, and at night she would wake with twitchings on the right side.

She continued work until she had to give up, chiefly on account of fatigue, and she returned to the Presbyterian Hospital on October 18. Soon speech became slurred and swallowing difficult, and weakness of the right side was added to the old parkinsonian rigidity. The visual fields were practically normal on October 21. The eyegrounds remained normal until about October 28; then papilledema rapidly developed bilaterally. Neither the lateral nor the anteroposterior roentgenogram of the head showed anything abnormal. The pineal shadow was in the midline. Spinal fluid obtained on October 18 was clear and colorless; the pressure was 190 mm.; the Wassermann and Lange reactions were negative, and the cell count was 1 per cubic millimeter. The protein measured 44 mg. and the sugar 68 mg. per hundred cubic centimeters. Chiefly in the hope that the severe headache might be relieved, burr holes were made in both occipital bones by Dr. Verbrugghen, on November 1. Abundant fluid, under increased pressure, was obtained from the right ventricle, but little came from the left. The protein content of the ventricular fluid was only 8 mg. per hundred cubic centimeters. No air was injected, as by this time my colleagues and I were convinced of the presence of an inoperable tumor on the left side. The relief from headache was complete and permanent, much to our surprise. The patient grew weaker and more stuporous from day to day, and finally fever, leukocytosis and signs of bronchopneumonia developed. She died on December 15. The clinical diagnosis was old, stationary paralysis agitans and a deep-seated cerebral tumor involving the left side of the brain stem. Necropsy revealed a large nodular tumor beneath the pons. It had eroded the bone between the sella and the foramen magnum, including the left posterior clinoid process. The pituitary gland was of normal size and appearance. The brain showed no other gross lesion. Examination of the rest of the body showed nothing of importance except beginning lobular pneumonia.

Histologic examination of the tumor revealed the structure of a typical chordoma. In one microscopic section the three characteristic stages were present: (1) the stage of densely packed cells, mostly without vacuoles, (2) the stage of extensive vacuole formation and (3) the stage of vacuole formation with the addition of syncytial formations. No mitotic figures were seen. As all the material had been placed in a solution of formaldehyde, it was impossible to use a special stain to determine whether the material in the vacuoles was glycogen, which is generally supposed to be present in this tumor. A piece of bone from the eroded basilar process which had been decalcified showed the same tumor structure.

At the time of this report nearly 100 cases of cranial chordoma have been reported, but a positive clinical diagnosis has been possible only by biopsy when the tumor extended into the nasopharynx, an occurrence which is said to have been noted in 20 per cent of cases. However, the diagnosis is almost certain if the roentgenogram reveals a depression in the bone near the spheno-occipital junction and if the ventriculogram shows elevation of the third and fourth ventricles above the base of the skull. The clinical picture is never completely convincing but is often extremely suggestive, and is dominated by progressive involvement of the cranial nerves. The commonest early local symptom is diplopia, due to paresis of the external rectus muscle, preceded or accompanied by localized or general headache. The other cranial nerves most frequently involved are the fifth and the tenth, but all the cranial nerves are subject to involvement, the selection depending on how far forward and backward the bone tumor extends. Penetration of the tumor into the brain is uncommon, and involvement of the cranial nerves is the result of their being stretched by the lifting up of the brain by the tumor at the base. Choked disk is not common and occurs late in the course of the disease. Hemiplegia and other symptoms of cerebral compression, if present, are of late appearance.

Surgical treatment has been tried in several cases, usually with such poor results that we do not regret our failure to attempt it in the present case. Even if the confusing preexisting paralysis agitans had not

delayed early recognition of the tumor, and if suitable roentgenologic examination had revealed its site, surgical intervention would probably have been futile.

The case here reported is typical of chordoma arising from the region of the clivus, the second most common location of this tumor, the most common site being the sacrococcygeal junction.

DISCUSSION

DR. BEN W. LICHTENSTEIN: Although it is easy to diagnose chordoma from the autopsy specimen, great difficulty may be encountered with small surgical specimens. The cuboidal cells of chordoma resemble so much the cells of hypernephroma and some varieties of carcinoma that one often thinks of a metastatic tumor at the base of the brain. The policy of fixing the entire specimen in a solution of formaldehyde continues, as in the past, even though such fixation precludes detailed histochemical analysis for glycogen and other substances.

DR. VICTOR E. GONDA: I should like to ask about the triplopia. It would be easy to understand the presence of this sign if the third, as well as the sixth, nerve had been involved. Otherwise, as has been taught, monocular diplopia is always "functional." If triplopia may be due to involvement of the sixth nerve, the present concept will have to be revised.

Were the false images seen to the same side, or were they crossed?

DR. LLOYD H. ZIEGLER: Perhaps my experience may throw light on the triplopia which Dr. Bassoe mentioned. Several years ago I saw a patient who had been drinking too much and who was disoriented and had visual and auditory hallucinations. He declared that real objects before him appeared double or triple and in the horizontal plane. Aside from tremor and disorientation, one of the chief signs was slow, jerky horizontal nystagmus. He said that the hallucinatory images of dogs, camels, cats and strange people, which he feared, also appeared double and triple and in the horizontal plane. Within a few days after his coming to the hospital, the true and false sensory experiences and the nystagmus disappeared. Defects in several extraocular muscles might produce two or three kinds of diplopia and images which were out of their ordinary position. Triplopia is rare in my experience and is not easy to understand, unless the conditions mentioned in this case (chiefly the slow nystagmus) may throw light on it.

DR. PETER BASSOE: I cannot answer Dr. Gonda's question. I asked Dr. E. V. L. Brown about it. He did not see the patient until the diplopia had passed away. He says there is such a thing as monocular diplopia, to which one may add triplopia due to involvement of the sixth nerve.

Influence of Estrogen and Androgen on the Chemical Constituents of the Brain. DR. ARTHUR WEIL.

Three years ago attempts to determine chemical differences in the lipid composition of the brain of the male and of the female white rat were reported on before this society (Influence of Thyroid and Sex Glands on the Chemical Constitution of the Brain, ARCH. NEUROL. & PSYCHIAT. **46**:547 [Sept.] 1941). It could be demonstrated then that at a given age (170 days) the phosphorus content of the alcoholic brain extracts, expressed as a percentage of the extract, was higher in the brain of the female rat than in that of the male rat. Further investigations disclosed that for young animals before puberty these values were about the same (2.70 per cent) for the two sexes; however, the decrease to the adult level, which begins approximately at the time of sexual maturity, occurred more abruptly in the male brain.

An analysis of the different lipid fractions suggested that this difference in phosphorus content was due to an increase of the phosphorus content of the cephalin fraction of the female brain (The Chemical Growth of the Brain of the White Rat and Its Relation to Sex, Growth **7**:257, 1943). Later I demonstrated, with Liebert (The Correlation Between Sex and Chemical Constitution of the Human Brain, *Quart. Bull., Northwestern Univ. M. School* **17**:117, 1943) that similar sexual differences existed in the human brain.

The final proof that such differences are due to hormonal influences, and not to chromosomal determination alone, was furnished by experimental injection of estrogen and androgen into normal and into gonadectomized rats. If the homologous glandular substance was injected into young rats before maturity, no effect on the chemical constituents of the brain could be observed. Injections of androsterone into young female rats (25 days old), however, produced the chemical characteristics of a male brain and, vice versa, the young male brain was transformed chemically into a female brain by injection of estrogen. Injections after puberty did not produce such clearcut reversals. Removal of the gonads before puberty, however, followed by injections of a preparation containing the hormone of the opposite sex, gave transformations similar to those produced in normal, immature animals. The accompanying tabulation illustrates these results. White rats about 60 days old were given injections for seventy days.

			Alcoholic Extract		Cephalin Fraction	
Animal	Body Weight, Gm.	Brain Weight, Mg.	% of Dry Brain	% Phosphorus	% of Precipitate	% Phosphorus
Male.....	352	1,880	31.8	2.21	45.1	2.28
Female....	223	1,795	32.2	2.35	53.4	2.43
Castrate, given estrogen *	275	1,865	30.7	2.23	43.0	2.45
Spayed given androgen *	294	1,805	32.9	2.22	46.4	2.21

* The estrogen given was estradiol benzoate; the androgen used was testosterone propionate. Both substances were supplied by Ciba Pharmaceutical Products, Inc.

Parasympathetic Regulation of High Potentials in the Electroencephalogram. DR. CHESTER W. DARROW and DR. WARREN S. MCCULLOCH and (by invitation) DR. JOHN R. GREEN, DR. EDWARD W. DAVIS and DR. HUGH W. GAROL.

The tendency for high potential slow waves appearing in the electroencephalogram during hyperventilation to be attended by an increase in heart rate suggested that the same autonomic mechanism might influence both the heart and the brain. It followed that if inhibition of vagal (parasympathetic) activity was the cause of the cardiac acceleration, section of parasympathetic pathways to the brain should increase high potential slow waves during hyperventilation.

Cats were operated on under ether anesthesia and then given beta erythroidine with artificial respiration. The parasympathetic pathways over the facial nerve, the geniculate ganglion and the greater superficial pet-

rosal and internal carotid nerves were interrupted by section of the facial or the greater superficial petrosal nerve. Two minute periods of hyperventilation, which had no effect on the electroencephalogram before section of the nerve, produced high potential slow waves after the nerve on one or on both sides was cut. Stimulation of the peripheral stumps of the nerves eliminated the slow waves. Intravenous administration of physostigmine likewise reduced or eliminated these waves, and atropine increased them. Potentials recorded from the greater superficial petrosal nerve were in part of the type shown by other investigators to occur during autonomic regulation by the carotid sinus.

The presence of a parasympathetic cholinergic mechanism which counteracts the spasmodic effects of hypocapnia on the cerebral blood vessels seems to be indicated.

DISCUSSION

DR. WARREN S. MCCULLOCH: Dr. Darrow commandeered one after another of the residents in brain surgery, and as each went to war, he took the next one. He and they did the work. This problem was a puzzling one. I had had an entirely different interpretation of the phenomenon in question, and I was more surprised than any one else at the outcome.

The principal place I filled was that of a "Doubting Thomas." My only contribution to the research was to insist that Dr. Darrow and his assistants actually demonstrate that the action potentials of the great superficial petrosal nerve diminished or disappeared prior to or during the appearance of the large slow brain waves induced by hyperventilation. This they did under my eyes. That, and only that, satisfied me as to the validity of the results.

But the society has a guest who knows far more about electroencephalograms than a tyro like me; I hope that Dr. Gibbs will have something to say about these observations.

DR. FREDERIC A. GIBBS: Dr. Darrow's recent paper (*Am. J. Physiol.* **140**:583 [Jan.] 1944) gave a hint of the extraordinarily interesting observations that he has reported here. Therefore, although his results differ from those which my own work would have led me to expect, I am neither shocked nor unduly surprised. Perhaps it is a kindness that he has not emphasized the extent of this divergence. My co-workers and I have considered the carbon dioxide level in the brain one of the primary determinants of cortical frequency, whereas Dr. Darrow is able to explain the slowing that he obtains without much regard to this factor.

Dr. Lennox, Mrs. Gibbs and I have reported that the cerebral carbon dioxide tension, which we believe is of crucial importance to cortical function, is protected by a homeostatic mechanism that in most normal persons is extraordinarily competent. This mechanism is based on the response of the cerebral blood vessels to variations in carbon dioxide content. When a person hyperventilates, the arterial carbon dioxide drops precipitously, but there is not a corresponding drop in cerebral carbon dioxide, as indicated by blood returning from the brain; cerebral blood vessels constrict when the arterial carbon dioxide falls, with the result that the carbon dioxide produced by the brain accumulates and compensates for the drop in carbon dioxide in the arterial stream. It appears that when this mechanism is competent, overventilation does not ordinarily result in the extreme slowing of cortical potentials with an associated increase in amplitude which we have referred to as a "big build-up." On the other hand, when this homeostatic mechanism is defective, that is, when the cerebral blood vessels do not constrict adequately, a notable build-up tends to occur. Therefore, the crucial factor under these circumstances appears to be the competence of the cerebral vasoconstrictor mechanism. However, Dr. Darrow reports that by interfering with this constriction and by favoring cerebral vasodilation he prevents the occurrence of a great build-up. As stated before, this is exactly the opposite of what we should have expected.

Dr. Darrow's results are of the greatest interest. They require that we scrutinize our own results and conduct further experiments. The question involved is of more than academic interest. High voltage slow waves are commonly precipitated by overventilation in epileptic patients, and it is likely that this response is closely related to the pathologic physiology of the convulsive disorders.

DR. CHESTER DARROW: In reply to Dr. Gibbs, I do not believe there is real conflict between our results and the beautiful demonstration by him and his collaborators that slow waves during hyperventilation may be due to deficient homeostatic regulation of the carbon dioxide level in the brain. In fact, in experiments which led up to the present one we found that the fall in blood pressure associated with hyperventilation might provide sufficient regulation of blood flow, and consequently of cerebral carbon dioxide, to prevent change in the electroencephalogram. Increased alpha potential and, presumably, cerebral vasoconstriction might occur when the blood pressure stopped falling, but high potential slow waves did not appear unless there was also pronounced cardiac acceleration. Only if there was a notable increase of heart rate, suggestive of inhibition of parasympathetic activity, did we generally find high potential slow waves due to hyperventilation. Thus our results, like those of Dr. Gibbs, suggest that effective regulation of the cerebral carbon dioxide content, whether by decrease in blood pressure or by cerebral vasoconstriction, is not accompanied by high potential slow waves. Results of the present experiment indicate that the combination of low carbon dioxide and reduced supply of cholinergic substances is the condition most provocative of such waves. It is likely that homeostatic vasoconstrictor regulation of the cerebral blood flow by carbon dioxide is most uniform and effective when acetylcholine is present to prevent local, spasmodic and excessive effects of hypocapnia.

Book Reviews

Recent Advances in Psychiatry. Special number of the *Journal of Mental Science,* published by authority of the Royal Medico-Psychological Association, January 1944. Volume 40. Price, 30 shillings. Pp. 509. London, J. & A. Churchill, Ltd.

This special number of the *Journal of Mental Science* is devoted to recent progress in psychiatry. An introduction by G. W. T. H. Fleming, managing editor, indicates that it is about fifteen years since Henry Devine issued his "Recent Advances in Psychiatry." The review covered in this volume is confined to work published during the five year period from 1938 to 1942, and in some instances the first half of 1943.

The authors hope to make this review a quinquennial publication. There are excellent surveys on problems of mental health services, genetics, anatomy of the nervous system, physiologic psychology and electroencephalography. A section is devoted to biochemistry of the nervous system, written by Derek Richter. Other reviews are concerned with vitamin deficiency and the psychoses, neuroendocrine relationships, mental testing, psychopathology and neuropathology.

W. Mayer-Gross and Norman P. Moore have written an excellent review of the problem of schizophrenia. Aubrey Lewis gives a commendable survey of the problem of depression. Psychopathic personality is considered in a separate chapter by Curran and Mallinson. W. H. Gillespie brings the problems of the psychoneuroses up to date, and H. Crichton-Miller and Grace Nicolle have a useful review of psychotherapy. Other chapters are devoted to arteriosclerotic, senile and presenile psychoses, the psychiatric aspects of head injury, neurosyphilis, mental deficiency and child psychiatry. Convulsion therapy, prefrontal leukotomy and insulin therapy are given exhaustive consideration, and W. Norwood East and P. K. McCowan each have an excellent chapter on delinquency and crime and the legal aspects of psychiatry respectively.

This volume is a timely review, much needed and well done, and each chapter has an excellent bibliography for purposes of reference. The book is highly recommended, and one hopes that the publication of such reviews will be continued in the present, useful form.

Personality and the Behavior Disorders: A Handbook Based on Experimental and Clinical Research. Edited by J. McV. Hunt, Associate Professor of Psychology, Brown University. Price, $10. Volumes I and II. Pp. 618; 619-1242, with illustrations. New York: The Ronald Press Co., 1944.

These two volumes are the best collection of articles yet published on personality and behavior disorders. The editor indicates that the study of personality and its development must draw heavily on all of the biologic sciences. These books bring together the work of forty contributors, to give the major theories, facts and clinical experiences from the sciences relating to development of personality. As a result of the growing importance of the work of Galton, Freud, Pavlov and others, many scientific data have been accumulated in this field. Each chapter is written by an expert, and there is a minimal amount of technical jargon.

Volume I contains chapters on theoretic approaches to personality, dynamic theories of personality, methods of assessing personality, behavior dynamics and biologic and organic determinants of personality. Volume II is concerned with experimental and sociologic determinants of personality. Sections are devoted to outstanding patterns of behavior disorders and to investigate correlates of behavior disorders, and a closing section is concerned with therapy and the prevention of behavior disorders.

Many sections are excellent—for example, "The Physiological Effects of Emotional Tension," by Leon J. Saul; "Experimental Analysis of Psychoanalytic Phenomena," by Robert Sears; "The Conditioned Reflex Method and Experimental Neurosis," by H. S. Liddell, and "Experimental Studies of Conflict," by Neil E. Miller. William H. Sheldon contributes an excellent chapter on "Constitutional Factors of Personality." Nathan W. Shack has a first class chapter on "Physiological Effects in Behavior," and Stanley Cobb writes the excellent chapter entitled "Personality as Affected by Lesions of the Brain." Each of these men is an expert in his respective field, and the presentation is clear, concise and to the point.

Volume II continues with an excellent chapter by Margaret Ribbel on "Infantile Experience in Relation to Personality Development," a chapter by Gregory Bateson on "Cultural Determinants of Personality" and a section by Leo Kanner on "Behavior Disorders of Childhood." Lawson Lowrey wrote the section entitled "Delinquent and Criminal Personalities." Warren Stearns covers the problem of unit personalities in the military services in a timely, well written article. The psychoneuroses are discussed by William Malamud and the functional psychoses by Norman Cameron. Paul W. Prue is the author of the chapter "Psychopathic Personalities," and William G. Lennox discusses seizure states. The editor, J. McV. Hunt, writes the chapter "Psychological Deficit," and Kenneth Appel and George S. Stevenson contribute to the section "Therapy and the Prevention of Behavior Disorders."

These contributions attempt to correlate the many aspects of behavior disorders. They fill a long-standing want in the field of psychology and its relations to clinical psychiatry. More validated studies of normal personality function are needed in order better to evaluate clinical problems. It is apparent that much useful information can come from careful study of man and infrahuman forms in the uncovering of the dynamic laws of personality development.

This work is a truly collaborative product and is highly recommended as one of the most useful symposiums on this subject. It should be read by every psychologist, sociologist and neuropsychiatrist.

The Origin and Function of Culture. By Géza Róheim, Ph.D. Nervous and Mental Disease Monograph Series, No. 69. Price, $2.50. Pp. 103. New York: Nervous and Mental Disease Monographs, 1944.

Róheim discusses his subject from a strictly freudian point of view. He carefully indicates in the foreword that he is cognizant of the many other theories of the origin of culture and civilization but that he is presenting his explanation from the point of view of Eros.

Róheim delineates the role that prolongation of immaturity in human beings plays in the development of

culture. He refers freely to his previous work in the field. He portrays the role of the medicine man in primitive society. The latter is frequently a psychotic or neurotic person, but one who has the capacity to utilize his peculiarities in such a way that he becomes a leader of his group. In addition, the author reports various libidinal situations that exist in certain primitive societies and traces their manifestations in the rituals, totems and taboos of the adults. He shows the direct relation of certain patterns of behavior in the group to libidinal situations created in the childhood of the members of these primitive societies. He briefly reviews the beginnings of the basic developments of civilization, such as agriculture. As an introduction to psychologic relations in primitive cultures the book is highly recommended. The reader should be aware that the book is not a volume of reference, but a brief presentation of the author's views and theories.

Manual of Psychological Medicine for Practitioners and Students. A. F. Tredgold. Price, $5. Pp. xi, plus 298. Baltimore: Williams & Wilkins Co., 1943.

The author of this volume is consulting physician to University College, London, and lecturer on mental deficiency at the London University. In his preface he states that the book is written because of the present need of general practitioners and students for information on psychiatric medicine. His desire is to create "a reasonably short, plain and practical account giving the essentials of this branch of medicine which would fill a very real wartime need." In this aim he has accomplished his purpose, for, in 298 pages, he has covered many of the essentials in the knowledge of practical psychiatry. The author has set down, both clearly and concisely, his concept of psychologic medicine in its entirety.

The usefulness of this book, however, is questionable. Like most compendiums, it covers too much ground, and too superficially. In the introduction, in which he discusses the "normal mind," theories of development and the darwinian concept of evolution are explained in elementary detail. The chapters dealing with the classification of "mental abnormality" and with general symptomatology are difficult to follow because there is no apparent theory behind the various classifications. Epilepsy is discussed under the heading of psychoneurosis, and Gowers is cited at great length, without any supplement from more recent work on either the neurophysiology or the clinical classifications of epilepsy. Similarly, in discussion of the major psychoses, one finds the concepts of Kraepelin and Bleuler, without mention of later investigators. The author differentiates sharply between the psychopathology of schizophrenia, a chronic hallucinatory psychosis, and dementia precox, a condition of primary mental decay.

Only 13 pages is devoted to therapy in any form, the major part of which is concerned with occupational therapy. Psychoanalysis is dealt with in 2 pages, in which the author's concept of the theories of Freud is set down and in which the results of psychoanalysis are stated to be questionable, impracticable and at times dangerous.

Finally, the major emphasis in the book is on the definitions of various forms of mental disease as designated by British law, and the various methods of commitment in England, neither of which can be of anything but academic interest to medical practitioners outside that country. To this reviewer, the book seems to contain an adequate presentation of the status of psychiatric practice several decades ago, and hence to be dangerous reading for the medical practitioner of today because of its omission of much that is at present important in the diagnosis and treatment of mental disease.

News and Comment

DIRECTORY OF MEDICAL SPECIALISTS CERTIFIED BY AMERICAN BOARDS 1945. PUBLISHED BY THE ADVISORY BOARD FOR MEDICAL SPECIALTIES

The biographic data of the first two editions of the "Directory of Medical Specialists" included only positions (internships, residencies or assistantships) held during the course of training of men up to the time of their certification by the American Boards, and positions on hospital and medical school staffs then currently held.

It is desired to extend these data in the third edition to include all formal appointments to hospitals and medical schools, with dates for which they were held, even though the appointee has now resigned, as well as records of all military service, including commissions and dates, whether in World War I, in the reserve forces in peacetime or in the present war.

Thus, a chronologically complete sketch of the entire career of a certified specialist is to be included in this third edition of the "Directory."

Membership or fellowship in national or sectional (not local) special societies and national general societies, with offices held and dates, should be reported.

Membership in recognized international medical societies may be included, but honorary or other membership in foreign medical societies should not be reported.

The certificate may refer to the second edition (1942) of the "Directory" for lists of medical societies to be included in his biographic sketch.

Families or secretaries of men absent in military service are asked to complete or correct previous listings or to prepare new forms now being mailed to physicians eligible for inclusion in the "Directory." Only persons certified by an official American Board can be included, and there is no charge for this listing.

This notice is published in response to many inquiries to assist physicians certified by the American Boards who are now engaged in correcting their previous listings or in preparing new sketches for the third edition of the "Directory," to be published early in 1945.

Communications should be addressed to the Directory of Medical Specialists, 919 North Michigan Avenue, Chicago 11.

NEW SOCIETY IN HABANA

The Sociedad Cubana de Neurologia y Psiquiatria has been founded in Habana. The officers for this year are as follows: president, Dr. Rodolfo J. Guiral; first vice president, Dr. Jesús Cornide; second vice president, Dr. Julio Reymondez; secretary, Dr. José A. Bustamante; vice secretary, Dr. Oscar Sagredo; treasurer, Dr. Luis Muñiz Angulo; vice treasurer, Dr. Rafael Larragoiti.